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CONTENTS OF PREVIOUS NUMBERS

SEPTEMBER, 1921. NUMBER 3

Demonstration of Food Protsins in Human Breast Milk by Anaphylactic Experiments on Guinea-Pigs. Their Probable Relationship to Certain Diseases of the Nursing Infant. A Preliminary Report. W. Ray Shannon, M.S., M.D., St. Paul.

Nutritional Keratomalacia in Infants, with a Report of Four Cases. S. G. Ross, M.D., Baltimore.

Medullary Malignancies of the Suprarenal Gland, with Report of Four Cases. William E. Carter, M.D., San Francisco.

Size and Weight in One Hundred and Thirty-Six Boarding School Boys (Groton). H. Gray, M.D., Boston and W. J. Jacomb, Groton, Mass.

Ideal Tables for Size and Weight of Private School Boys. H. Gray, M.D., Boston.

A Bacteriologic Study of the Fecal Flora of Infants and Children (The Lack of Association of Nutritional Disorders with a So-Called "Putrefactive" Intestinal Flora). Wilburt C. Davison, M.D., and L. V. Rosenthal, M.D., Baltimore.

Congenital Heart Block. Paul D. White, M.D., and Richard S. Eustis, M.D., Boston, and William J. Kerr, M.D., San Francisco.

Clinical Department:
Peritonitis as a Complication of Scarlet Fever. Ethel C. Dunham, M.D., New Haven, Conn.

Progress in Pediatrics:
Review of Literature for 1920 on Neurology of Children. Lewis J. Pollock, M.D., Chicago.

Book Review.

OCTOBER, 1921. NUMBER 4

Metabolism Studies in Infants Suffering from Chronic Nutritional Disturbances (Athrepsia). Kirsten Utheim, M.D., St. Louis.

On the Etiology of Hemorrhagic Disease of the New-Born. C. F. Gelston, M.D., San Francisco.

Severe Infantile Malnutrition. The Energy Metabolism with the Report of a New Series of Cases. Fritz B. Talbot, M.D., Boston.

The Food Requirements of Children. II.—Protein Requirement. L. Emmett Holt, M.D., and Helen L. Fales, New York.

Observations on the Capillaries of the Normal Infant. Katherine M. Mayer, Chicago.

Clinical Investigations on Orthostatic Albuminuria. Hideo Saito, M.D., Nagasaki, Japan.

A Study of the Incidence of Hereditary Syphilis. P. C. Jeans, M.D., and J. V. Cooke, M.D., St. Louis.

Clinical Department:
Calcification of the Skin in a Child. John Lovett Morse, A.M., M.D., Boston.

Progress in Pediatrics:
Review of the Literature of 1920 on Genito-Urinary Conditions in Children. Lewis Webb Hill, M.D., Boston.

NOVEMBER, 1921. NUMBER 5

Observations on Infantile Tetany. B. Kramer, F. F. Tisdall, and J. Howland, Baltimore.

Experimental Albuminuria in the Rabbit. Hideo Saito, M.D., and Kumataro Gondo, Nagasaki, Japan.

The Vital Capacity of the Lungs and Its Relation to Exercise Tolerance in Children with Heart Disease. Standards for Normal Vital Capacity for Children. The Lung Capacity in Certain Intrathoracic Conditions. May G. Wilson, M.D., and Dayton J. Edwards, Ph.D., New York.

Studies of Infant Feeding. XIV. Chemical Studies of Certain Dry Milk Products Used in Infant Feeding. Alfred W. Bosworth, Boston.

A Clinical and Radiographic Study of the Thymus in Infants. Kenneth D. Blackfan and Karl Little, Cincinnati.

The Size of the Infantile Palate. B. S. Denzer, M.D., New York.

Congenital Goiter. Report of Four Cases. William B. Porter, M.D., and R. A. Vonderlehr, M.D., Richmond, Va.

The Etiology and Treatment of Ammonia Dermatitis of the Gluteal Region of Infants. J. V. Cooke, M.D., St. Louis.

The Abdominal Pain of Throat Infections. Joseph Brennemann, M.D., Chicago.

Some Records Regarding Absences and Their Causes in the Lincoln School of Teachers College. Howard H. Mason, New York.

Rare Cardiac Anomaly. Cor Triloculare Biventriculare in Mirror Picture Dextrocardia with Persistent Omphalomesenteric Bay, Right Aortic Arch and Pulmonary Artery Forming Descending Aorta. Bret Ratner, M.D., New York, Maude F. Abbott, M.D., and W. W. Beattie, M.D., Montreal, Canada.

Clinical Department:
A Case of Tuberculous Meningitis with a Dry Spinal Subarachnoid Space Due to Diffuse Tubercle Infiltration of the Spinal Meninges. Joseph C. Regan, M.D., and G. W. Holmes Cheney, M.D., Brooklyn.
Intermitting Fever in a Child Due to Mastoiditis. Diagnosis by Positive Roentgen-Ray Findings. Jerome S. Leopold, M.D., New York.

DECEMBER, 1921. NUMBER 6

Blood Studies in the New-Born. Morphological; Chemical; Coagulation; Urobilin and Bilirubin. William Palmer Lucas, M.D.; Bradford French Dearing, M.D.; Hal R. Hoobler, M.D. Anita Cox; Martha R. Jones, Ph.D., and Francis Scott Smyth, M.A., San Francisco.

The Clinical Significance of Calcium Concentration in the Serum of Children and Possible Errors in Its Determination. B. Kramer, F. F. Tisdall and J. Howland, Baltimore.

Genital Tuberculosis in Male Children. J. Dellinger Barney, M.D., Boston.

Traumatic Diaphragmatic Hernia in a Girl of Eight Years of Age. Murray B. Gordon, M.D., and Daniel L. Golann, M.D., Brooklyn.

Iso-Agglutinins in the Blood of the New-Born. Basil B. Jones, M.D., Boston.

Isohemolysins in Human Blood, with Especial Reference to the Blood of the New-Born. Basil B. Jones, M.D., Boston.

Modes of Infection in Pyelitis. Henry F. Helmholz, M.D., Rochester, Minn.

Studies on Infant Feeding. XV. The Calcium of Cow's Milk in Its Relation to the Digestion and Absorption of Casein. Protein Curds in Stools. Alfred W. Bosworth, Boston.

Book Review.

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CONTENTS OF VOLUME 23

JANUARY, 1922, NUMBER 1 PAGE

THE NATURE OF THE PLANTAR REFLEX IN EARLY LIFE AND THE CAUSES OF ITS VARIATION. W. M. FELDMAN, M.D., B.S., LONDON..... 1

CAN YEAST BE USED AS A SOURCE OF THE ANTINEURITIC VITAMIN IN INFANT FEEDING? AMY L. DANIELS, PH.D., IOWA CITY..... 41

THE INCIDENCE OF PROTEIN SENSITIZATION IN THE NORMAL CHILD. M. MURRAY PESHKIN, M.D., AND WILLIAM L. ROST, M.D., NEW YORK 51

AN UNUSUAL EXANTHEM OCCURRING IN INFANTS. ROY M. GREENTHAL, M.D., ANN ARBOR, MICH..... 63

THE RESPIRATORY EXCHANGE IN A CASE OF BILIARY ATRESIA. G. B. FLEMING, GLASGOW, SCOTLAND..... 66

BACTERIOLOGIC STUDIES OF ONE HUNDRED AND SIXTY-FIVE CASES OF PNEUMONIA AND POSTPNEUMONIC EMPYEMA IN INFANTS AND CHILDREN. ARTHUR BATES LYON, M.D., BOSTON..... 72

CLINICAL DEPARTMENT:

CONGENITAL TUBERCULOSIS WITH REPORT OF CASE. GEORGE P. PRATT, B.SC., M.D., OMAHA..... 88

FEBRUARY, 1922. NUMBER 2

IS THERE MORE THAN ONE KIND OF RICKETS? P. G. SHIPLEY, BALTIMORE; E. A. PARK, NEW HAVEN, CONN.; E. V. MCCOLLUM AND NINA SIMMONDS, BALTIMORE 91

THE VALUE OF THE ROUTINE USE OF THE COLLOIDAL GOLD REACTION IN ACUTE EPIDEMIC POLIOMYELITIS. JOSEPH C. REGAN, M.D., AND G. W. HOLMES CHENEY, M.D., NEW YORK..... 107

THE CLINICAL VALUE OF INTRAPERITONEAL INJECTIONS OF SALT SOLUTION. J. CLAXTON GITTINGS, M.D., AND JOHN D. DONNELLY, M.D., PHILADELPHIA 124

HEMATOMAS IN THE HEART VALVES OF CALVES. LAURA FLORENCE, PH.D., PRINCETON, N. J..... 132

EFFECT OF TONSILLECTOMY ON NUTRITION IN TWELVE HUNDRED CHILDREN. ALBERT D. KAISER, M.D., ROCHESTER, N. Y..... 139

CLINICAL DEPARTMENT:

REPORT OF A CASE OF GANGRENE OF THE FEET FOLLOWING DIPHTHERIA. MURRAY B. GORDON, M.D., AND BENJAMIN NEWMAN, M.D., BROOKLYN 142

PROGRESS IN PEDIATRICS:

RÉSUMÉ OF LITERATURE (1920) ON TUBERCULOSIS IN CHILDREN. MAY MICHAEL, M.D., CHICAGO..... 146

MARCH, 1922. NUMBER 3

CHRONIC DIFFUSE NEPHRITIS IN CHILDHOOD. REPORT OF A CASE WITH REVIEW OF THE LITERATURE. CARL H. GREENE, ROCHESTER, MINN.. 183

THE ANTISCORBUTIC PROPERTY OF FRUITS. II. AN EXPERIMENTAL STUDY OF APPLES AND BANANAS. MAURICE H. GIVENS, H. B. MCCLUGAGE AND E. G. VAN HORNE, ROCHESTER, N. Y..... 210

INDICES OF THE STATE OF NUTRITION IN CHILDREN. H. GRAY, M.D., AND G. H. EDMANDS, BOSTON..... 226

RESULTS FOLLOWING THE ADMINISTRATION OF ALKALI PHOSPHATES TO SPASMOPHILIC, RACHITIC AND NORMAL INFANTS. JOSEPH K. CALVIN, S.B., M.D., AND MAXWELL P. BOROVSKY, S.B., M.D., CHICAGO..... 238

STUDIES OF INFANT FEEDING. XVI. A BACTERIOLOGIC STUDY OF THE FECES AND THE FOOD OF NORMAL BABIES RECEIVING BREAST MILK. ELSIE W. BROWN AND ALFRED W. BOSWORTH, BOSTON..... 243

CLINICAL DEPARTMENT:

A CASE OF ALKAPTONURIA IN AN INFANT. J. B. BILDERBACK, M.D., PORTLAND, ORE. 259

PROGRESS IN PEDIATRICS:

REVIEW OF THE LITERATURE ON RESPIRATORY DISEASES FROM JANUARY, 1920, TO JUNE, 1921. PHILIP MOEN STIMSON, M.D., NEW YORK CITY (TO BE CONTINUED)..... 261

APRIL, 1922. NUMBER 4

INDICATIONS FOR THE REMOVAL OF THE SPLEEN IN INFANTS AND CHILDREN. FREDERIC H. BARTLETT, M.D., NEW YORK CITY..... 283

THE BACTERIOLOGY OF THE NORMAL INFANT'S URINE. HENRY F. HELMHOLZ, M.D., AND FRANCES MILLIKIN, ROCHESTER, MINN..... 309

416790

APRIL, 1922—Continued PAGE

THE VALUE OF CUTANEOUS SENSITIZATION TESTS EMPLOYED IN ECZEMA AND PAPULAR URTICARIA OF CHILDHOOD. D. M. SIDLICK, M.D., AND FRANK CROZER KNOWLES, M.D., PHILADELPHIA..... 316
STUDIES OF INFANT FEEDING. XVII. A BACTERIOCHEMICAL STUDY OF THE ACID STOOLS EXCRETED BY BREAST-FED AND BOTTLE-FED INFANTS. ALFRED W. BOSWORTH, H. KENNETH WILDER, MARGUERITE E. BLANCHARD, ELSIE W. BROWN AND MARION F. MCCANN, BOSTON... 323
PROGRESS IN PEDIATRICS:
REVIEW OF THE LITERATURE ON RESPIRATORY DISEASES FROM JANUARY, 1920, TO JUNE, 1921. PHILIP MOEN STIMSON, M. D., NEW YORK CITY (CONCLUDED)..... 338

MAY, 1922. NUMBER 5

NEPHRITIS IN CHILDREN. GLADYS BOYD, M.B. (TOR.), TORONTO..... 375
ECZEMA IN BREAST-FED INFANTS AS A RESULT OF SENSITIZATION TO FOODS IN THE MOTHER'S DIETARY. W. RAY SHANNON, M.S., M.D., ST. PAUL 392
SITTING-HEIGHT AND STEM-LENGTH IN PRIVATE SCHOOL BOYS. H. GRAY, M.D., BOSTON 406
THE PRESENCE OF FORMIC ACID IN THE URINE OF INFANTS AND OLDER CHILDREN. MORLEY D. MCNEAL, M.D., ROCHESTER, MINN., AND CHARLES J. ELDRIDGE, M.D., CHICAGO..... 419
THE PROPHYLACTIC AND THERAPEUTIC VALUE OF PERTUSSIS VACCINE. G. R. DAVIES, M.D., SYRACUSE, N. Y..... 423
THE CHLORIN CONTENT OF COW'S AND GOAT'S MILK AND FORMULAS COMMONLY USED IN INFANT FEEDING. WARREN R. SISSON, M.D., AND W. DENIS, PH.D., BOSTON..... 431
CLINICAL DEPARTMENT:
RESPIRATORY OBSTRUCTION RESULTING IN DEATH. CASE REPORT WITH NECROPSY FINDINGS. JAMES W. BRUSE, M.D., AND STUART GRAVES, M.D., LOUISVILLE, KY..... 438
PROGRESS IN PEDIATRICS:
A REVIEW OF THE LITERATURE OF SYPHILIS IN INFANCY AND CHILDHOOD. PARK J. WHITE, M.D., ST. LOUIS..... 442
BOOK REVIEWS 470

JUNE, 1922. NUMBER 6

THE FOOD REQUIREMENTS OF CHILDREN. III. FAT REQUIREMENT. L. EMMETT HOLT, M.D., AND HELEN L. FALES, NEW YORK CITY.... 471
THE FREQUENCY OF TUBERCULOSIS IN CHILDHOOD. FRANZ HAMBURGER, STYRIA, AUSTRIA 481
IDIOPATHIC APLASTIC ANEMIA. REPORT OF CASE IN A CHILD FOUR YEARS OLD; MARKED IMPROVEMENT FROM TRANSFUSION. CHARLES HERRMAN, M.D., NEW YORK CITY..... 484
SPASMOPHILIA AND THE ALKALI RESERVE OF THE BLOOD. JOSEPH K. CALVIN, S.B., M.D., AND MAXWELL P. BOROVSKY, S.B., M.D., CHICAGO 493
ACTIVE IMMUNIZATION OF NURSES AGAINST DIPHTHERIA IN A CHILDREN'S HOSPITAL. J. V. COOKE, M.D., ST. LOUIS..... 496
THE ASPIRATION OF STEARATE OF ZINC IN INFANCY. A CLINICAL AND EXPERIMENTAL STUDY. HENRY HEIMAN, M.D., AND PAUL W. ASCHNER, M.D., NEW YORK CITY..... 503
HEALING OF HYPERTROPHIC PYLORIC STENOSIS AFTER THE FREDET-RAMMSTEDT OPERATION. MARTHA WOLLSTEIN, M.D., NEW YORK... 511
THE VITAMIN REQUIREMENT OF VARIOUS SPECIES OF ANIMALS. III. THE PRODUCTION AND CURE OF XEROPHTHALMIA IN THE SUCKLING. VICTOR E. NELSON, ALVIN R. LAMB AND V. G. HELLER, AMES, IOWA..... 518
A PECULIAR ERUPTIVE DISEASE OCCURRING IN INFANCY. JAMES H. PARK, JR., M.D., AND JEFFREY C. MICHAEL, M.D., HOUSTON, TEXAS..... 521
THE VALUE OF THE INTRACUTANEOUS TUBERCULIN TEST IN EXTENSIVE TUBERCULOSIS. WILLIAM H. HAPP AND HORTON R. CASPARIS, BALTIMORE 527
THE BACTERIOLYSANT THERAPY OF BACILLARY DYSENTERY IN CHILDREN. THERAPEUTIC APPLICATION OF BACTERIOLYSANTS; D'HERELLE'S PHENOMENON. WILBUR C. DAVISON, M.D., BALTIMORE..... 531
PROGRESS IN PEDIATRICS:
A REVIEW OF THE LITERATURE OF SYPHILIS IN INFANCY AND CHILDHOOD. PARK J. WHITE, M.D., ST. LOUIS (CONCLUDED FROM PAGE 469) 535

American Journal of Diseases of Children

Vol. 23 JANUARY, 1922 No. 1

THE NATURE OF THE PLANTAR REFLEX IN EARLY LIFE AND THE CAUSES OF ITS VARIATIONS*

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LONDON

INTRODUCTORY AND HISTORICAL

In 1898 Babinski [1] called attention to the plantar reflex as a diagnostic sign in lesions of the pyramidal tracts of the spinal cord. The reflex which he called *le phenoméne des orteils* consists, as is now so well known, of the plantar flexion of the big toe consequent on the tickling of the inner border of the sole of the foot. This is the normal response. When the pyramidal tracts are diseased in any part of their course, the same stimulus applied to the sole of the foot results in a dorsiflexion instead of a plantar flexion of the toe. Since then this peculiarity of the toe reflex in organic lesions of the pyramidal tract has become known as Babinski's sign or the Babinski phenomenon, and has been a subject of investigation by many observers all over the world, including Buzzard,[2] Collier,[3] Cohn,[4] Crocq,[5] Cestan and Le Sourd,[6] Van Epps,[7] Van Gehuchten,[8] Glorieux,[9] Guidiceandrea,[10]

* Received for publication Aug. 13, 1921.

* Thesis approved for the degree of Doctor of Medicine in the University of London.

1. Babinski, J.: Du phènomène des orteils et de sa valeur semiologique. Semaine méd. 321, 1898; Sur la transfermation du regime des réflexes cutaines, etc., Rev. neurol. 58, 1904.

2. Buzzard, T.: On the differential diagnosis of insular sclerosis from hysteria, B. M. J. **1**:1077, 1899.

3. Collier, J.: An Investigation of the Plantar Reflex, etc., Brain **22**:71, 1899.

4. Cohn, M.: Ueber die Bed. der Zehenreflex, Neurol. Centralbl. **18**:580, 1899.

5. Crocq. J.: Die Reflexe Mit Rücksicht auf die Lebensversicherung, Wien. med. Wchnschr. **52**: Nos. 17-25, 1902.

6. Cestan and Le Sourd: Contribution a l'etude du phenomène des orteils, Gaz. d. hôp. 1249, 1899.

7. Epps. C. Van: The Babinski reflex, J. Nerv. & Ment. Dis. 214, 1901.

8. Van Gehuchten: Quoted by Passini.[21]

9. Glorieux: Quoted by Passini.[21]

10. Guidiceandrea: Quoted by Passini.[21]

Kalischer,[11] König,[12] Munch-Petersen,[13] Okada,[14] Schüler,[15] Schneider,[16] Yoshimura [17] and many others. The various findings are not in agreement. Thus, while all French and British observers, as well as most investigators of other countries, confirm Babinski's statement, other neuropathologists (Cohn, Kornilow,[18] Schüler, Tumpowski,[19] and others) either consider the test unreliable, or attach no diagnostic significance to it. Kornilow says:

> Although Babinski's sign often occurs in the presence of functional disturbance of the pyramidal tract, there are also cases of disease of the pyramidal tracts without the presence of Babinski's sign. On the other hand, cases have been observed in which there was an undoubted Babinski's sign, and which at the necropsy revealed perfectly normal pyramidal tracts. The differential diagnostic significance of the Babinski phenomenon for the purpose of distinguishing organic from hysterical hemiplegias is not pathognomonic. There are cases of hysteria in which the Babinski sign is present.

Chodz'ko [20] criticizes the observations of Babinski's opponents as inexact and as probably due to defective methods of observation.

While, therefore, there is no absolute unanimity on the matter, the balance of evidence is preponderatingly, one might even say, overwhelmingly in favor of the diagnostic significance of the Babinski phenomenon.

Passini [21] confirms Babinski's statement in the case of children suffering with pyramidal lesions (cerebral diplegia and monoplegia, congenital hydrocephalus with spastic paresis of the lower extremities, spinal diseases, e. g., compression myelitis due to spinal caries).

Babinski having found this peculiarity of the toe reflex in pyramidal disease of the spinal cord, expected a priori to find similar dorsiflexion of the big toe as a physiologic phenomenon in early infancy, at a time

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11. Kalischer, O.: Ueber den Normalen und Pathologischen Zehenreflex, Virchows Arch. f. path. Anat. 1899.
12. König, W.: Ueber die bei Reizung der Fussohle zu beobachtenden Reflexerscheinungen, etc., Berl. klin. Wchnschr. 822, 1899.
13. Munch-Petersen, H.: Die Hautreflexe und ihre Nervenbahnen, Deutsch. Ztschr. f. Nervenh. **22**:1902. Quoted by Yoshimura.[17]
14. Okada, E.: Ueber das Babinskische Zehenphänomen, Neurologia **1**:13, 1902. Quoted by Yoshimura.[17]
15. Schüler, L.: Beobachtungen uber Zehenreflexe, Neurol. Centralbl. **19**: 585, 1899.
16. Schneider, H.: Ueber das Zehenphänomen Babinski's, Berl. klin. Wchnschr., 1901.
17. Yoshimura, K.: Ueber das Babinskische Phänomen, Mittheilungen aus den Medizinischen Fakultät der Kaiserlichen Universität zu Tokyo **8**:213, 1909.
18. Kornilow, A.: Ueber Cerebrale und Spinale Reflexe, Deutsch. Ztschr. f. Nervenh. **23**: 1903.
19. Tumpowski, A.: Ueber den diagnostischen Werth des Babinskischen Phänomens, Neurol. Centralbl. **20**:663, 1901; abstr. from Medycyna (Polish).
20. Chodz'ko: Beitrag zur Bed. des Babinskischen phänomens, Abstr. from Gaz. lekarska, 1901, in Neurol. Centralbl. **20**:663, 1901.
21. Passini, F.: Ueber den Normalen Grosszehenreflex bei Kindern, Wien. klin. Wchnschr. 952, 1900.

when the pyramidal tracts are not yet fully developed. When he came to test for this phenomenon in new-born infants, he found that his surmise was correct. Indeed, he believes that dorsiflexion of the toe is the normal plantar reflex in all children who have not yet learned to walk, but that when the child is able to walk the character of the reflex changes from dorsiflexion to plantar flexion. Cestan and Le Sourd found in all infants under 1 year old a dorsiflexion, but they failed to find the exact age at which the reflex changes from dorsiflexion to plantar flexion.

Collier found the age of transition in normal healthy children to be between 2 and 3 years. He says:

> I have met with the typical adult response in children 2 years old who were particularly forward and "strong on their legs." In weakly and especially in rickety children, the infantile response may be as late as the age of 4 years.

During sleep, Collier finds the flexor response (i. e., plantar flexion) to be the rule even in infants, but in some children, even up to the age of 12 years, who showed plantar flexion when awake, there was dorsiflexion during sleep. Collier, like Babinski, associates this dorsiflexion of the toe in infants with imperfect myelination of the pyramidal system, and calls attention to the fact that in monkeys (in whom the pyramidal tracts are incompletely developed) the plantar reflex is dorsiflexor in type.

Schüler examined 100 children and found the following results:

No reflex response	in 40 per cent.
Dorsiflexion	in 30 per cent.
Plantar flexion	in 12 per cent.
Indefinite response	in 18 per cent

The 40 per cent. in which there was no response were all young infants. He concludes that in normally developed infants plantar flexion is the prevailing response during the fourth quarter of the first year but that the nature of the response varies with the general development of the infant. Thus an infant aged 14 months whose weight was only that of one aged 5 months, showed dorsiflexion, while a 5 months' old infant which was well developed showed plantar flexion.

Cohn, like Schüler, finds no response at all in about half the cases examined.

Gallewski [21a] set himself the task of discovering whether the Babinski reflex, whose clinical significance in adults cannot be doubted, is also to be correlated with the degree of development of the pyramidal tracts in infants. Utilizing the Weigert-Pal's method of staining

21*a*. Gallewski, M.: Histologische und klinische Utersuchungen über die Pyramidenbahn und das Babinskische Phänomen im Säuglings Alter. Dissertation Breslau 1902 (British Museum).

as modified by Kulschitzky, he finds that there is a definite parallelism between myelination and function in the pyramidal system as measured by the presence or absence of Babinski's sign. He examined nine infants between birth and 8 months of age and found that in all of them, except one (case 6) the reflex was dorsiflexor in type and they all showed greater or less poverty in myelin in the pyramidal regions (the degree of myelination varying with the age of the infant).

One child (Case 6) presented an extreme atrophy (only weighing 2,070 gm., i. e., about 4½ pounds), but the reflex was plantar flexor in character. The pyramidal tracts, both crossed and direct, were very poorly myelinated so that microscopically they resembled the picture shown in the case of an infant one month old.

Here, then, is a case which apparently militates against Babinski's theory. In order, however, to bring the case into line with that theory, Gallewski calls to his aid Kalischer's explanation of the Babinski phenomenon. Kalischer believes that those muscles which are less well innervated will respond reflexly in preference to an antagonistic group of muscles whose innervation is better. In infants in whom the foot is still, atavistically, a prehensile rather than a progressional organ, the interossei are a stronger group of muscles than the extensors of the toes, and, therefore, as a result of reflex irritation the extensor hallucis responds. When the foot definitely becomes an organ of locomotion, the relative strength of the antagonistic groups of muscles becomes reversed and dorsiflexion is replaced by plantar flexion. In Case 6, which was one of extreme atrophy, Gallewski makes the unwarranted assumption that the interossei were more atrophied than the extensors, and thus plantar flexion is obtained.

Walton and Paul [22] find no characteristic reflex in infants. Morse,[23] in an investigation of 254 infants not suffering from any nervous disease found no plantar reflex at all in ninety (35 per cent.); reflex absent on one side and plantar flexion on the other in eighteen (7 per cent.); reflex absent on one side and dorsiflexion on the other in fifteen (6 per cent.); plantar flexion on both sides in sixty-four (25.5 per cent.); dorsiflexion on both sides in fifty-four (21.5 per cent.); plantar flexion on one side and dorsiflexion on the other in thirteen (5 per cent.).

He found further that during the first nine months bilateral dorsiflexion was more frequent than plantar flexion, while after that age the condition was reserved. His findings are shown in Table 1.

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22. Walton and Paul: Contribution to the Study of the Plantar Reflex, etc., J. Nerv. & Ment. Dis. **27**:305, 1900.

23. Morse, J. L.: A Study of the Plantar Reflex in Infancy, Pediatrics **11**:13, 1901.

Morse conclues that (1) there is no constant plantar reflex in the first year; (2) there is no constant plantar reflex in the second year, although it approximates that of the adult; (3) the normal reflex is not established at 2 years.

Engstler [23a] examined a large number (eighty-nine) of healthy infants [24] in Graz and found (mostly in the sleeping state) that (1) in premature and mature infants at birth, there was a typical dorsiflexion of the big toe in all cases; (2) during the first year 93 per cent. had dorsiflexion and only 7 per cent. plantar flexion, (3) between the second and third years, plantar flexion is 95 per cent.; (4) at the end of the first year, 50 per cent. had dorsiflexion and 50 per cent. had plantar flexion; (5) Babinski's sign becomes an abnormal phenomenon after the second year.

TABLE 1.—MORSE'S FINDINGS AS TO REFLEXES IN NORMAL INFANTS *

	Percentages First Year (200 Cases)	Percentages Second Year (54 Cases)
Plantar flexion on one side and dorsiflexion on the other	5 (8.5)	5.5 (6.5)
Plantar flexion on one side and no reflex on other	7 (11.8)	7.5 (8.7)
Dorsiflexion on one side and no reflex on other	6 (10.2)	5.5 (6.5)
No reflex on either side	41	15.0
Plantar flexion on both sides	16.5 (28)	57.5 (67.4)
Dorsiflexion on both sides	24.5 (41.5)	9 (10.9)

* The percentages in parentheses are those calculated after deducting the eighty-two cases in the first year and the eight cases in the second year which gave no reflex on either side.

Griffith [25] accepts Engstler and Leri's [26] statement that "the plantar reflex in the new-born is characterized by dorsal flexion of the toes" and that "gradually this condition changes, but it is not till the third year that plantar flexion is the rule."

Barnes [27] agrees with Kalischer that the change from dorsiflexion to plantar flexion occurs at the beginning of walking even if the latter is delayed to the age of 4 or 5 years; but he finds that the response in infants is not so strong as in severe spastic states in adults—proving that even at birth the pyramidal system is by no means functionless, although myelination of fibers has not occurred.

Leri finds in 166 infants aged from 1 day to 3 years that (1) at birth dorsiflexion is the practically general rule (in this way agreeing with Muggia's findings but disagreeing with Guidiceandrea). (2)

23*a*. Engstler, G.: Ueber den Fussohlenreflex, etc., Wien klin. Wchnschr. **18**:567, 1905.

24. The total number of cases he examined was 1,000 but 109 gave no response.

25. Griffith, J. P. C.: Diseases of Infants and Children, 1919.

26. Leri, A.: Le reflexe des orteils chez les enfants, Rev. neurol. 689, 1903.

27. Barnes S.: The Diagnostic value of the plantar reflex, Rev. neurol. & psych. 352, 1904.

After 3 years plantar flexion is the rule, although dorsiflexion may occur without any pathologic significance. (3) Between 1 and 3 years dorsiflexion is not met with, as a rule, except (a) in affections of the central nervous system; but in these cases it has not the same significance as in adults because the pyramidal tracts soon after their formation are still more susceptible to every slight irritation especially of a toxic character; (b) in severe disturbances of nutrition which produce a retardation of development of the pyramidal tracts. (4) The change from dorsi flexion to plantar flexion occurs in normal infants at about 5 or 6 months. (5) Plantar flexion is of more diagnostic significance than dorsiflexion since in children in whom walking starts late it remains the only sign that the pyramidal tract is properly developed and that there is no fear in them of Little's disease.

Finizio [28] studied the reflex in 500 new-born infants during the first three days of life. He found no reflex in 5 per cent.; indecisive reflex in 10 per cent.; dorsiflexion in 15 per cent.; plantar flexion in 70 per cent. Finizio also studied the modified plantar reflexes (Schaffer's and Brissaud's reflexes) and found both of them present at birth.

As regards the effect of labor on the character of the reflex, Finizio finds that in difficult forceps cases there is dorsiflexion—possibly because in such cases some meningeal hemorrhage is produced. In true Little's disease, however, he finds plantar flexion. Sometimes in forceps cases he found a brisk knee-jerk with a Babinski phenomenon on one side, and plantar flexion on the other, possibly from inequality of the pressure of forceps on the fetal skull.

Furmann [29] who investigated 502 infants and children found the plantar reflex absent only in twenty-four cases. He further found that in infants under 9 months old dorsiflexion of the toe is almost the rule, while the transition from dorsiflexion to plantar flexion occurs between 9 months and 2 years. According to this observer, the occurrence of a Babinski phenomenon after the age of 2 years is indicative of an affection of the pyramidal tracts.

Laurent [30] found in 110 children between a few hours and 3 years old a constant Babinski phenomenon during the first six months, and one alternating with a normal plantar reflex between 6 months and 15 months. After that age an extensor (dorsiflexor) response is, according to him, very rare, except pathologically.

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28. Finizio, G.: Le Reflex de la plante du pied chez les Nouveaux, XIII Cong. Internat. de Med., Compt. Rend. Sect. med. de l'enfance; Paris, 1900, p. 399.

29. Furmann: Die Reflexe des Säuglings, Dissertation St. Petersburg, 1903; abstr. by Gundobin in Die Besonderheiten des Kindesalters, Berlin, 1912.

30. Laurent, R.: Evolution des reflexes chez l'enfant, These de Toulouse, 1905; abstr. in Brit. J. Child. Dis. 460, 1906.

OBJECTS OF THE PRESENT INVESTIGATION

As there are wide divergences in the findings of the nature of the plantar reflex in infants and the age at which the reflex becomes definitely of the adult type, I have investigated about 500 children from birth (including premature births) up to the age of 8 years, with the following objects:

1. To test the truth of the generally accepted statements (a) that dorsiflexion is the prevailing phenomenon during infancy; (b) regarding the dependence of the nature of the plantar reflex on the state of the development of the pyramidal tracts as found postmortem.

2. The conformation of the postmortem findings with theoretical expectations.

3. To find the correlation (if any) between the response and various developmental and environmental states.

4. Localization of the reflexogenetic zone in infants.

Nature of the Material.—The material on which this research has been carried out consisted mainly of infants brought to the outpatient department of the Infants Hospital. Some observations, however (especially those at birth), have been made in private cases as well as in a few instances in cases at the Chelsea Infirmary. For the last I am indebted to my cousin, Dr. Victor Feldman, assistant medical officer at that institution, for having kindly allowed me to make the observation.

Method of Investigation.—In all cases, the observations were made when the feet were warm, i. e., after they (as well as the infant) had been well wrapped up in a blanket. The stimulus was applied by means of the thumb nail. In some cases, in order to warm the feet, it was necessary to sponge them with warm water. In a case in which I assisted at the birth of the child, I had the opportunity in a transverse presentation to test the reflex intra-uterinally by enclosing the ankle between ring and middle fingers, stimulating the sole of the foot with the thumb and feeling the direction of the toe with the index finger. In the case of older infants it was often found necessary to pacify them by means of the breast or the bottle before testing for the reflex in order to avoid the error between a true Babinski phenomenon and the movements of the toes and foot which often occur as a defensive mechanism against the irritation and which is not unlike the Babinski reflex. The plantar defense reaction consists in rapid dorsiflexion of the foot with simultaneous withdrawal of the foot from the source of irritation, with or without extension of the big toe. In a true Babinski phenomenon the great toe extends slowly. In some cases the reflex was tested during sleep. It often happened that there was, at first, no response of the toes at all to the plantar stimulus, but frequent repetition of the stimulus very rarely failed, although it did sometimes fail, to elicit some sort of a response.

I have no exact record of the number of cases at every age period which failed to give a response, but as the total number of cases examined was about 500 and of these 426 gave some sort of response, the total percentage of absent reflexes was about 15.

Results.—In the following tables I have classified my results in 426 cases from birth onward in which a response was obtainable. From these it is seen that:

1. During the first year, the proportion between flexor and extensor responses is approximately 4:1 and that in approximately 16 per cent. the reflex is variable, being either different in the two sides or different on each side at different times.

2. During the second year, the proportion between flexor and extensor responses is approximately 3:1 and in about 14 per cent. the reflex is variable.

3. During the third year, in 81 per cent. of cases the response was plantar flexor in type, and in 19 per cent. it was variable. There were no cases with a constant bilateral Babinski phenomenon.

4. During the fourth year the proportion between flexor and extensor responses was 14:1, and in 5 per cent. of cases it was variable.

5. After the fourth year, there was only one case (Case 617) in which there was an extensor response on one side, in all the remaining eleven cases the response was invariably plantar flexor in type on both sides. I have, however, notes of one case, a boy, aged 9 years and 4 months, in which the reflex was the typical Babinski phenomenon (Case 753). This boy was brought to me because he walked on tiptoe. There was no history of obstetric difficulties at birth. He began to walk and talk at the normal periods, but he walked on his toes from the beginning. There is a history of several miscarriages by his mother both before and after the birth of this child, and some time before this child was born the mother passed through a typical spurious pregnancy and labor.

On examining the child I found him to be a bright, intelligent boy. His weight was 6 stone (84 pounds) and his height 4 feet 6 inches. He preferred to walk on his toes, but was also able to walk on the soles of his feet normally. His knee jerks as well as the other reflexes were not increased, and there was no ankle clonus, but the plantar reflex was typically dorsiflexor in type. The flexors of the leg, however, were tense. It is possible, therefore, that this is a mild case of Little's disease, although there is no other evidence to substantiate the diagnosis.

I have summarized the findings of various observers, but it must be noted that the groups of cases are not strictly comparable as regards age.

TABLE 2.—Data on Flexion by Various Observers

	Engstler (891 cases)	Schüler (60 cases)	Morse (194 cases)	Finizio (500 new-born)	Feldman (426 cases)
Plantar flexion.....	26.7%	20%	39%	73%	67.0%
Dorsi flexion.....	73.3%	50%	33%	16%	16.4%
Indefinite.....	.....	30%	28%	11%	16.6%

These figures have been calculated after deducting the numbers of cases, in each series, in which the reflex was absent.

Age at Which the Sign Disappears.—While it would be impossible to say definitely that dorsiflexion never occurs after the sixth year in a child whose pyramidal tracts are not diseased, I have been unable to elicit it in any one of a considerable number of cases in which I have looked for it after that age. Case 777 is the only one among a dozen or more cases of children of that age in which there was dorsiflexion of the toe on one side (right). On the other hand, Case 618 was a normal girl, 4 years old, who had marked dorsiflexion of the toe seven hours after a convulsive seizure.

Correlation Between Response and the State of Development of the Pyramidal Tracts as Found Postmortem.—In two cases in which I had records of the nature of the plantar response I had an opportunity of examining sections of the spinal cord postmortem, after keeping in Müller's fluid for six weeks, and staining sections by Weigert-Pal's method.

Case 1 (128).—This was a case of congenital deformity. Both tibiae were curved. The fibulae were absent and no definite tarsal bones could be made out radiographically. The feet were practically suspended from the legs in the same straight line. The infant was extremely wasted, having weighed only 5 pounds 3 ounces at one month, and about the same weight at death at the age of 7 weeks. The plantar reflex was absent on the right side and plantar flexor in type on the left side throughout its life. It died at the age of 7 weeks. The cord showed the presence of some myelinated fibers in all the white columns and in each region (cervical, dorsal and lumbar), although myelination was incomplete in the crossed pyramidal tracts in the lower region. There was no noticeable difference in the state of myelination of the two sides of the cord, in spite of the difference in response. As, however, the response was altogether absent on the right side, the fault could not rest in the pyramidal tract but in the lower neuron. Moreover, the anterior horn cells being developed early in the fetus, the fault must have been in the peripheral nerves. The brain was of good size and showed no abnormality. There was only one kidney which was horseshoe shaped, although there were two suprarenals.

Case 2 (614).—This was a male child whose weight at birth was 8 pounds. The plantar reflex was flexor (i. e., plantar flexion) on both sides at the age of 14 hours, and flexor on one side and extensor (i. e., dorsiflexion) on the other at 28 hours. It died suddenly at the age of 35 hours. Postmortem the thymus was found to be 11.6 gm. in weight (i. e., about normal). The spinal cord showed some myelination of all the white columns although the myelination was more incomplete in the crossed pyramidal tracts. There is no obvious difference in the degree of myelination between the two sides of the cord, in spite of the difference in the character of the response on the two sides.

Fig. 1.—Photomicrograph of section of the dorsal cord region of the spinal cord from Case 614; × 14.

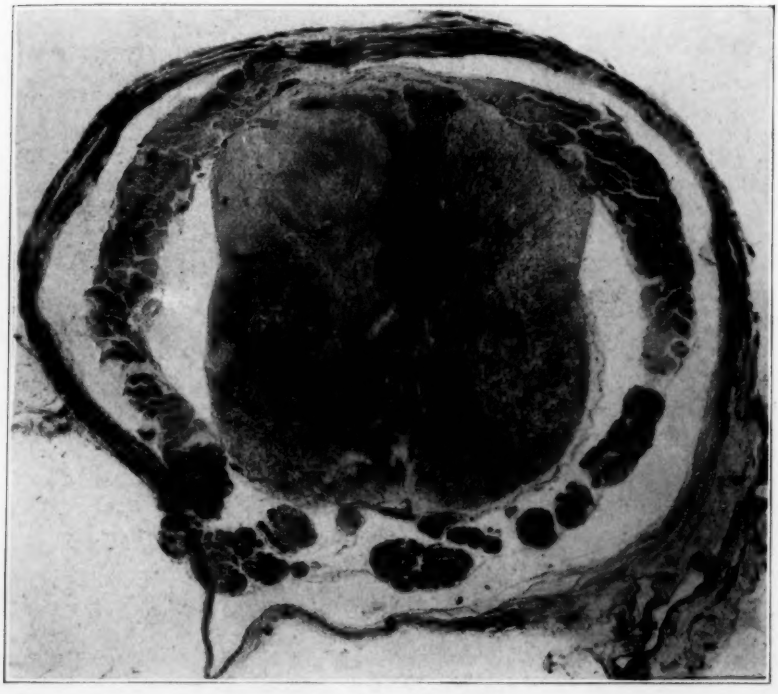

Fig. 2.—Photomicrograph of section of dorsal region of the spinal cord from Case 128; × 14.

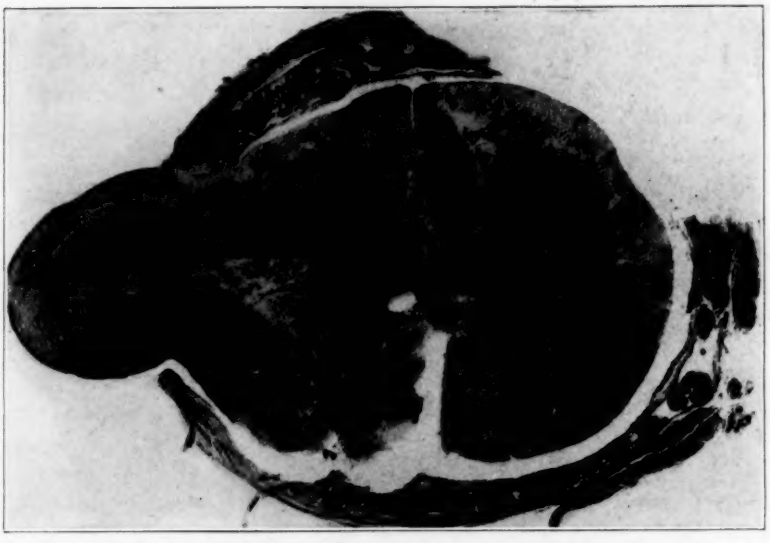

Fig. 3.—Photomicrograph of section of lumbar region of spinal cord from Case 614; × 14. The cord was injured during its removal.

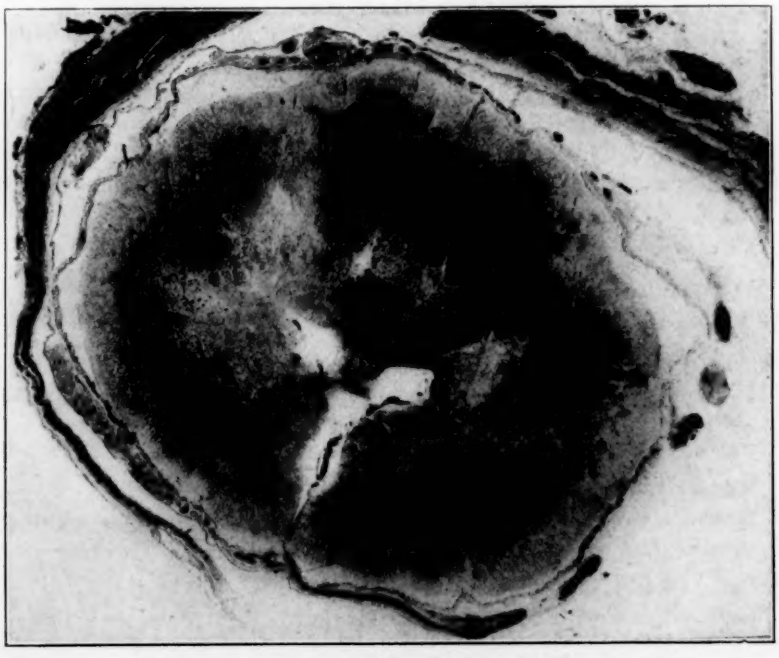

Fig. 4.—Photomicrograph of lumbar region of spinal cord from Case 128; × 14.

On comparing the cords of Cases 128 and 614 there is obviously a greater poverty of myelin in Case 128 (the microphotographs do not show it so well as the slides examined by means of a projector), and yet in this case there was no dorsiflexion on either side although in Case 614 there was plantar flexion on the right side.

Hence, it appears that the degree of myelination of the pyramidal tracts in early infancy is not sufficient to explain the nature of the plantar response.

Moreover, the occurrence of a true Babinski phenomenon either on one side or on both sides after the age of 3 years in normally developed children, in whom according to all authorities the pyramidal tracts are fully developed, points to the same conclusion.

The inference that one is led to draw from these facts is that although the pyramidal tracts are not completely developed at birth, there is a sufficiency of myelination in them to give a normal flexor plantar response; but (as I shall show later) that in virtue of the greater susceptibility of children to reflex disturbances in the circulatory system, the pyramidal tracts may be compressed and relaxed alternately as the result of such circulatory disturbances.

What Factors, if Any, Influence the Nature of the Plantar Response in Infants?—Sex: The first factor that would occur to one is the influence of sex. I find that in very early infancy a normal flexor response is slightly more common in girls than in boys, but this may be accounted for by the paucity of numbers. If we take, however, all cases during the first six months, the first year, the first two years, etc., I find that there is an equality of plantar flexor responses in both sexes, although the extensor or dorsiflexor response is at all periods more common in girls than in boys to the extent of about 6 or 8 per cent (see Tables 4, 6, 8).

2. Prematurity (Table 5): The effect of prematurity is shown quite clearly in Table 5.[31] (a) During the first month no less than six out of eight cases showed a Babinski phenomenon on each side, one only a bilateral plantar flexion, and the other case showed plantar flexion on one side and dorsiflexion on the other. (b) After the fifth week the effect of prematurity begins to pass off, although if the prematurity is very great, its effect is more lasting (Case 738, at the age of 7 months). Throughout the first year thirteen out of thirty-eight infants prematurely born had a positive Babinski phenomenon (34 per cent.). (c) The E : F ratio which is 6 : 1 during the first month becomes 1 : 4 during the second month and 1 : 6 during the third. It still keeps on falling during the second quarter but remains stationary after that.

31. See especially the E : F rates in Table 5, which is very instructive.

TABLE 3.—INCIDENCE OF THE VARIOUS RESPONSES AT DIFFERENT AGES FROM BIRTH ONWARDS

Age	No. of Cases	F	E	V		No. of Cases	F	E	V
At birth.....	5	3	1	1					
First day.....	3	2	1	1					
Second day..	1	..	..	1	First week.....	12	7	3	2
Third day....	1	1	..	..					
Fifth day....	2	1	1	..					
1-2 weeks.....	2	1	1	..					
2 weeks.....	8	4	3	1	Second week.....	10	5	4	1
2-3 weeks.....	4	3	1	..					
3 weeks.....	6	3	1	2	Second half of month.	22	14	5	3
4 weeks.....	12	8	3	1					
					First month.....	44	26 (59%)	12 (27.3%)	6 (13.7%)
5 weeks.....	14	10	2	2					
6 weeks.....	18	15	2	1	Second month.....	79	63 (80%)	7 (8.8%)	9 (10.2%)
7 weeks.....	13	9	..	4					
8 weeks.....	34	29	3	2	Total first 2 mos.	123	89 (72.4%)	19 (15.4%)	15 (12.2%)
9 weeks.....	6	3	1	2					
10 weeks.....	17	10	6	1	Third month.....	72	39 (54.2%)	23 (32%)	10 (14.8%)
11 weeks.....	7	2	4	1					
12 weeks.....	42	24	12	6	Total first quarter...	195	128 (65.6%)	42 (21.5%)	25 (12.9%)
14 weeks.....	8	6	..	2					
4 months....	30	17	8	5	Second quarter.....	80	55 (68.8%)	9 (11.2%)	16 (20%)
5 months....	19	13	1	5					
6 months....	23	19	..	4	Total first half year..	275	183 (66.5%)	51 (18.5%)	41 (15%)
7 months....	16	12	1	3					
8 months....	13	7	1	5	Third quarter.....	38	25 (66%)	5 (13%)	8 (21%)
9 months....	9	6	3	..					
					Total first 9 mos.	313	208 (66.4%)	56 (17.9%)	49 (15.7%)
10 months....	8	8	..	..					
11 months....	10	6	1	3	Last quarter.....	23	16 (70%)	2 (8.7%)	5 (21.3%)
12 months....	5	2	1	2					
					Total first year.....	336	224 (66.7%)	58 (17.3%)	54 (16%)
1 yr. 1 mo. ...	12	4	5	3					
1 yr. 2 mos. ..	6	4	1	1	First quarter 2d year..	22	12	6	4
1 yr. 3 mos. ..	4	4	..	..					
1 yr. 4 mos. ..	3	3	..	..					
1 yr. 5 mos. ..	3	..	1	2	Second quarter 2d yr..	12	8	2	2
1 yr. 6 mos. ..	6	5	1	..					
					Total 1st half 2d year	34	20 (59%)	8 (23.5%)	6 (17.5%)
1 yr. 7 mos. ..	3	2	..	1					
1 yr. 8 mos. ..	3	3	..	..	Third quarter 2d yr. ..	10	8	1	1
1 yr. 9 mos. ..	4	3	1	..					
1 yr. 10 mos.	3	1	2	..					
1 yr. 11 mos.	8	2	..	1	Fourth quarter 2d yr.	12	9	2	1
2 years.....	6	6	..	..					
					Total 2d half 2d yr. ..	22	17 (72.7%)	3 (18.2%)	2 (9%)
					Total whole of 2d yr.	56	37 (66%)	11 (20%)	8 (14%)
					Total whole of first 2 years.....	392	261 (66%)	69 (17%)	62 (16%)
2-2½ years...	7	6	..	1					
2½-3 years...	9	8	..	1	Third year.....	16	13 (81%)	..	2 (19%)
3-4 years.....	5	3	1	1	Fourth year.....	5	3	1	1
					Total first 4 yrs.	413	277 (67%)	70 (17%)	65 (16%)
4-9 years.....	13	11	0	2	.....	13	11	..	2
					Total first 9 yrs.	426	288 (67.6%)	70 (16.4%)	67 (16%)

TABLE 4.—Influence of Sex on the Nature of the Plantar Reflex

Age	Males				Females			
	No. of Cases	F	E	V	No. of Cases	F	E	V
0	2	1	..	1	3	2	1	..
First day	2	1	1	..	1	1	..	..
Second day	1	..	..	1	0	..	..	..
Third day	0	..	..	..	1	1	..	..
5 days	2	1	1	..	0	..	..	..
First week	7	3	2	2	5	4	1	..
1-2 weeks	0	..	..	..	2	1	1	..
2 weeks	2	..	2	..	6	4	1	1
Second week	2	..	2	..	8	5	2	1
2-3 weeks	4	3	1	..	0	..	..	..
3 weeks	6	3	1	2	0	..	..	..
Third week	10	6	2	2	0	..	..	..
4 weeks	9	7	1	1	2	1	1	..
First month	28	16 (57%)	7 (25%)	5 (18%)	15	10 (67%)	4 (27%)	1 (6%)
5 weeks	8	6	..	2	6	4	2	..
6 weeks	6	5	..	1	10	8	2	..
7 weeks	7	4	..	3	6	5	..	1
8 weeks	16	13	1	2	18	16	2	..
Second month	37	28 (75.7%)	1 (2.6%)	8 (21.7%)	40	33 (82.5%)	6 (15%)	1 (2.5%)
9 weeks	2	1	..	1	3	2	1	..
10 weeks	9	5	3	1	8	5	3	..
11 weeks	6	2	3	1	1	..	1	..
12 weeks	27	16	7	4	15	8	5	2
Third month	44	24 (54.5%)	13 (30%)	7 (15.5%)	27	15 (55.5%)	10 (37%)	2 (7.5%)
Total first 3 months	109	68 (62.5%)	21 (19.2%)	20 (18.2%)	82	58 (70.7%)	20 (24.4%)	4 (4.9%)
14 weeks	6	5	..	1	2	1	..	1
4 months	17	11	3	3	13	6	5	2
5 months	8	5	..	3	10	7	2	1
6 months	15	13	..	2	7	5	..	2
Second quarter	46	34 (74%)	3 (6.5%)	9 (19.5%)	32	19 (60%)	7 (22%)	6 (18%)
Total first half year	155	102 (65.8%)	24 (15.5%)	29 (18.7%)	114	77 (67.6%)	27 (23.7%)	10 (8.7%)
7 months	11	8	1	2	5	4	..	1
8 months	6	3	..	3	7	4	1	2
9 months	8	5	3	..	1	1	..	..
Third quarter	25	16 (64%)	4 (16%)	5 (20%)	13	9 (70%)	1 (7.7%)	3 (22.3%)
10 months	2	2	..	..	6	6	..	..
11 months	6	3	1	2	3	2	..	1
12 months	1	1	..	..	1	1	..	..
Fourth quarter	9	6 (66.6%)	1 (1.1%)	2 (2.2%)	10	9 (90%)	..	1 (10%)
Total second half of first year	34	22 (64.7%)	5 (14.7%)	7 (20.6%)	23	18 (78.3%)	1 (4.4%)	4 (17.3%)
Total whole of first year	189	124 (65.6%)	29 (15.4%)	36 (19%)	137	95 (70%)	28 (20%)	14 (10%)
Second year	26	20 (77%)	2 (7.7%)	4 (15.3%)	28	16 (57%)	9 (32.2%)	3 (10.8%)
Total first 2 years	215	144 (67%)	31 (14.4%)	40 (18.6%)	165	111 (67.3%)	37 (22.4%)	17 (11.3%)
Third and fourth years	11	9	..	2	9	5	1	3
Total first 4 years	226	153 (67.6%)	31 (13.7%)	42 (18.7%)	174	116 (66.6%)	38 (21.8%)	20 (11.6%)
Cases over 4 years	8	5	1	2	6	6	..	..
Total	234	158 (67.5%)	32 (13.5%)	44 (19%)	180	122 (67.8%)	38 (21.5%)	20 (11.1%)

3. Degree of Development of the Infant as Determined by Length and Weight (Tables 6 and 7): It has been stated by a number of observers, e. g. Leri, Schüler and others that the Babinski phenomenon is more common in poorly developed than in well developed infants. I have therefore analyzed my cases according to their weights as well as according to their lengths.

TABLE 5.—EFFECT OF PREMATURITY

Age	No. of Cases	F	E	V	E:F Ratio
First week	3	1	2	..	
2 weeks	2	..	1	1	
3 weeks	1	..	1	..	
4 weeks	2	..	2	..	
First month	8	1 (12.5%)	6 (75%)	1 (12.5%)	6:1
5 weeks	1	..	1	1	
6 weeks	1	1	..	..	
8 weeks	3	3	..	..	
Second month	5	4 (80%)	1 (20%)	..	1:4
9 weeks	1	..	..	1	
10 weeks	2	2	..	..	
11 weeks	2	..	1	1	
12 weeks	4	4	..	..	
Third month	9	6 (66.6%)	1 (11.1%)	2 (22.2%)	1:6
First 3 months	22	11 (50%)	8 (36.3%)	3 (13.7%)	7:10
4 months	3	1	2	..	
5 months	1	1	..	..	
6 months	3	3	..	..	
Second quarter	7	5 (71%)	2 (29%)	..	4:10
Total first 6 months	29	16 (55.3%)	10 (34.5%)	3 (10%)	
7 months	3	1	1	1	
8 months	1	1	..	..	
9 months	3	1	2	..	
10 months	2	2	..	..	
11 and 12 months	..	..	..	..	
Total second 6 months	9	5 (55.5%)	3 (33.3%)	1 (11.1%)	6:10
Total first year	38	21 (55.2%)	13 (34.2%)	4 (10.5%)	6:10

Weight: It is difficult to say exactly what weight at any particular age can be considered abnormally low, but I think it will be agreed that infants under 5 pounds during the first week, under 6 pounds at a month old, under 7¼ pounds at 2 months, under 8½ pounds at 3 months, under 11 pounds at 6 months and under 16 pounds at 1 year are considerably under weight. In the tables I have classified the infants according to these arbitrary standards, and I have also indicated what I considered below normal at intermediate ages. The detailed results are shown in the tables (Table 6), but dividing the cases into three monthly periods we obtain the following figures:

(a) During the first three months, there was a total of 133 cases of more or less normal development out of which ninety (67.7 per cent.) showed a normal (i. e., plantar flexion) reflex; twenty-five (18.8 per

TABLE 6.—INFLUENCE OF STATE OF DEVELOPMENT OF INFANT AS JUDGED FROM ITS WEIGHT

Age	Well Developed				Under Normal Development				
	No. of Cases	F	E	V	No. of Cases	F	E	V	Pounds
0	4	3	0	1	1	0	1	..	
First day	3	2	1	..	..	..	..	..	
Second day	1	..	..	1	..	..	..	..	
3 days	1	1	..	..	..	..	..	..	
5 days	..	..	..	..	2	1	1	0	
First week	9	6	1	2	3	1	2	0	Under 5
Second week	1	1	..	..	1	..	1	..	}
2 weeks	5	4	1	..	3	1	2	1	Under 5½
3 weeks	4	2	0	2	2	1	1	..	Under 5¼
4 weeks	9	7	2	0	3	1	1	1	Under 6
Total first month	28	20	4	4	12	4	7	2	
5 weeks	10	7	1	2	4	3	1	..	Under 6½
6 weeks	13	10	2	1	2	2	..	..	Under 6¾
7 weeks	10	6	..	4	3	2	..	1	Under 7
8 weeks	28	23	3	2	6	6	..	..	Under 7¼
Total first 2 mos.	89	66	10	13	27	17	9	3	
9 weeks	3	1	1	1	3	2	..	1	Under 7½
10 weeks	10	4	5	1	7	6	1	..	Under 8
11 weeks	2	..	2	..	5	2	2	1	Under 8¼
12 weeks	25	16	6	3	15	8	4	3	Under 8½
Total first 3 mos.	129	87 (67.4%)	24 (18.5%)	18 (14.1%)	57	35 (60%)	15 (26%)	8 (14%)	
3½ months	5	4	..	1	3	2	..	1	Under 8¾
4 months	22	14	5	3	7	3	2	2	Under 9
5 months	13	11	1	1	6	2	1	3	Under 10
6 months	13	10	..	3	8	7	..	1	Under 11
Total second 3 mos.	53	39 (73.6%)	6 (11.3%)	8 (15.1%)	24	14 (58.3%)	3 (12.5%)	7 (29.2%)	
Total first 6 mos.	182	126 (69.2%)	30 (16.1%)	26 (14.7%)	81	49 (70%)	18 (22%)	15 (18%)	
7 months	10	9	1	..	6	3	..	3	Under 12
8 months	7	5	..	2	5	2	..	3	Under 13
9 months	2	1	1	..	7	5	2	..	Under 14
10 months	3	3	..	..	4	4	..	..	Under 14½
11 months	8	4	1	3	2	2	..	..	Under 15
12 months	4	1	1	2	1	1	..	..	Under 16
Total second 6 mos.	34	23 (67.6%)	4 (11.7%)	7 (20.6%)	25	17 (68%)	2 (8%)	6 (24%)	
Total first year	216	149 (70%)	34 (16%)	33 (14%)	106	64 (61.3%)	20 (18.8%)	21 (19.8%)	
First half 2d year	12	6	4	2	21	4	3	..	Under 17½ at 13 mo. under 20 at 18 mo.
Second half of 2d year	7	5	1	1	9	9	..	..	Under 21 at 19 mo. under 23 at 2 yr.
Total second year	19	11 (58%)	5 (20%)	3 (16%)	30	23 (76.6%)	4 (30.3%)	3 (10%)	

cent.) showed a Babinski phenomenon and the remaining eighteen (13.5 per cent.) had a reflex of a variable character. Of the fifty-seven cases definitely under standard weight, thirty-four (60 per cent)

showed a plantar reflex of the normal adult type, fifteen (26 per cent.) presented a Babinski sign, and eight (14 per cent.) had a variable reflex.

(b) During the first six months there is again a greater percentage of Babinski phenomena in poorly nourished than in well nourished infants (22.2 per cent. against 16.6 per cent.).

(c) During the first year there is still a preponderance of Babinski signs in favor of badly nourished infants, but this preponderance is not so great as during the earlier periods (19 per cent. as against 16 per cent.).

TABLE 7.—INFLUENCE OF STATE OF DEVELOPMENT AS JUDGED FROM LENGTH

Age	Well Developed				Under Normal Development				
	No. of Cases	F	E	V	No. of Cases	F	E	V	Inches
0-4 weeks	13	10	2	1	7	4	2	1	Under 19
1-2 months	58	44	6	8	16	14	1	1	Under 20
2-3 months	52	29	16	7	17	9	5	3	Under 20¼
Total first 3 mo.	123	83 (67.4%)	24 (19.5%)	16 (13%)	40	27 (67.5%)	8 (20%)	5 (12.5%)	
3-4 months	29	19	6	4	6	2	1	3	Under 20½
5 months	16	12	..	4	2	1	1	..	Under 21
6 months	17	13	..	4	4	4	..	..	Under 22
Second quarter	62	44 (71%)	6 (9.6%)	12 (19.2%)	12	7 (58.3%)	2 (16.6%)	3 (25%)	
Total first half year	185	127 (69%)	30 (16%)	28 (15%)	52	34 (65%)	10 (19%)	8 (15.5%)	
7 months	10	9	1	..	5	2	..	3	Under 23
8 months	11	6	5	..	1	1	..	..	Under 23½
9 months	6	4	2	..	3	2	1	..	Under 24
Third quarter	27	19	8	..	9	5	1	3	
10 months	5	5	..	..	1	1	..	..	Under 24
11 months	9	5	1	3	..	..	..	..	Under 24½
12 months	3	2	1	..	..	..	..	..	Under 25
Last quarter	17	12	1	4	1	1	..	..	
Total second 6 mos.	44	31 (70%)	9 (20%)	4 (10%)	10	6 (60%)	1 (10%)	3 (30%)	
Total first year	229	158 (69%)	39 (17%)	32 (14%)	62	40 (64.5%)	11 (17.7%)	11 (17.7%)	

From these facts it would seem, therefore, that malnutrition is a factor which operates against the production of a normal plantar flexor response. On analyzing the cases further, however, it is seen that such a conclusion is erroneous. We find that:

(d) During the period from 4 to 6 months there is a practically equal percentage of Babinski signs in the two groups of cases (12.5 per cent. in underdeveloped and 11.3 per cent. in well developed infants), while during the second semester there is actually a greater percentage of Babinski phenomena in normally developed infants than in infants below the standard (11.7 and 8 per cent, respectively).

(e) The same is the case during the period from 1 to 2 years (the percentage of Babinski phenomena in the normal and below normal groups of cases being 26 and 13.5 per cent., respectively).

It is, therefore, clear that malnutrition per se has no influence on the nature of the plantar response, but that inasmuch as during the early months prematurity and malnutrition go hand in hand, it is probably the prematurity and not the malnutrition that is responsible for the greater presence of Babinski phenomena in the undernourished infants. Indeed, if we subtract from the fifty-seven infants under normal weight during the first three months the seventeen which owed their defective weight to prematurity, and out of which seven had a plantar flexor response, seven a true Babinski sign and three a variable response, we obtain the following figures:

Total number of under weight cases, forty; normal adult type of response, twenty-seven (67.5 per cent.); Babinski sign in eight (20 per cent.); variable reflex in five (12.5 per cent.), which are almost identically the same percentages as those prevailing amongst the normally developed infants.

Length: It is generally recognized that the length of an infant is a far better criterion of growth than is weight.[32] I have, therefore, arranged my cases at each age period in two groups, viz., one in which the infants are approximately of the average length at that age and another in which the infants are considerably shorter than the average (Table 7). I have indicated in the tables the lengths which I consider to be definitely below the normal. We there see that

(a) During the first month, 77 per cent. of the normal cases (i. e., ten out of thirteen) had a normal plantar reflex, 15 per cent. (i. e., two out of thirteen) had a Babinski and 7½ per cent. gave a variable response, while of the seven cases under normal length at the same period 57 per cent. had a normal plantar reflex, 28.5 per cent. gave a Babinski phenomenon and 14.25 per cent. gave a variable response.

(b) During the first three months, out of 123 normal cases and forty cases below the normal length, the percentages of cases of normal, Babinski and variable reflexes were respectively identical in the two groups, viz., 67.5, 20 and 13 per cent.

(c) During the second quarter, there was a great percentage preponderance of Babinski phenomena in the case of infants below the standard length, but this may be accounted for by the paucity of the number of cases in the abnormal group.

(d) For the period from birth (0) to 6 months, the preponderance in the abnormal group was very small (19 as against 16 per cent.).

(e) During the third quarter there was a percentage preponderance of Babinski phenomena in favor of the normal group of cases but this again may be accounted for by the very small number of cases in the abnormal group.

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32. Feldman, W. M.: The Principles of Antenatal and Postnatal Child Physiology Pure and Applied, London, 1920.

(f) For the period from birth (0) to 12 months, the respective percentages in the two groups were practically identical.

It is, therefore, reasonable to infer that just as in the case of weight, length in itself has no influence on the nature of the plantar reflex, and that the preponderance of Babinski phenomena during the first month was due to the prematurity which was the cause of deficient length.

Cephalic Index (i. e. $\frac{\text{Width of head}}{\text{Length of head}} \times 100$) (Tables 8 and 9).

A head which has an index of less than 79 is dolichocephalic or long-headed. An index of between 79 and 81 makes the head mesocephalic and one of over 81 makes it brachycephalic. In spite of the plasticity of the infant's head, it has been asserted by Boas [33] and Ripley [34] (and my own observations tend to confirm it) that after the neonatal period, when the effects of the obstetric molding on the infant's head has passed off, although the cephalic index slightly decreases with increasing age, yet the type of the head remains the same, i. e., a brachycephalic head will always remain brachycephalic, and so forth. Now, contrary to the opinion held by most anthropologists that the cephalic index is a racial character determined by heredity, Boas believes that the shape of the head is a character which is affected by environment—including intellectual activity, brachycephaly being the accompaniment of marked activity of the brain. Therefore, I though it would be of some academic interest to investigate the influence of the cephalic index on the plantar reflex because if Boas' contention is true, and if further it is true, as it almost certainly is, that the cephalic index is unaffected by growth, then one ought to find that in cases of brachycephaly the plantar reflex should be more commonly of the normal adult type, while the Babinski phenomenon should be more frequently associated with a dolichocephalic head. In the statistics presented (Tables 8 and 9), I have, in order to avoid error, excluded all cases of infants under 6 weeks old in whom the shape of the head might still be undergoing change.

The following are the results:

1. During the first quarter, the incidences of the different responses for the three different shapes of head are almost respectively identical.

2. During the first half year the percentages of normal responses for the three different varieties of head are practically identical, and while the incidence of the Babinski phenomenon is practically the same for dolichocephalic and mesocephalic heads, that in the case of brachycephalic heads is perceptibly less than in the other two (viz., 10.5 per cent. as compared with about 17 per cent.).

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33. Boas, F.: The Form of the Head as Influenced by Growth, Am. Science, n.s. 50; Changes in Bodily Forms of Descendants of Immigrants, Washington, 1910.

34. Ripley, W. Z.: American Science, n. s. **111**:888, 889.

TABLE 8.—Influence of Shape of Head on the Nature of the Plantar Reflex

Age	Dolichocephaly				Mesocephaly				Brachycephaly			
	No. of Cases	F	E	V	No. of Cases	F	E	V	No. of Cases	F	E	V
7 weeks	5	3	..	2	2	2	0	..	2	2	..	..
8 weeks	12	12	..	..	11	10	1	..	4	2	..	2
Second month	17	15	..	2	13	12	1	..	6	4	..	2
9 weeks	5	3	1	1	1	..	..	1	..	..	..	..
10 weeks	8	4	4	..	3	1	2	..	3	2	1	..
11 weeks	4	2	2	..	..	..	..	..	..	..	..	..
12 weeks	22	15	4	3	7	4	2	1	7	3	2	2
Third month	39	24	11	4	11	5	4	2	10	5	3	2
Total first quarter	56	39 (70%)	11 (20%)	6 (10%)	24	17 (71%)	5 (21%)	2 (8%)	16	9 (56%)	3 (19%)	4 (25%)
14 weeks	4	4	..	..	..	..	..	..	1	1	..	..
4 months	8	4	2	2	9	5	2	2	6	4	1	1
Fourth month	12	8	2	2	9	5	2	2	7	5	1	1
5 months	8	6	1	1	3	1	..	2	6	4	..	2
6 months	5	4	..	1	5	4	..	1	9	7	..	2
Second quarter	25	18 (72%)	3 (12%)	4 (16%)	17	10 (58.8%)	2 (11.7%)	5 (29.5%)	22	16 (72.7%)	1 (4.6%)	5 (22.7%)
Total first 6 months	81	57 (70%)	14 (17.3%)	10 (12.6%)	41	27 (66%)	7 (17%)	7 (17%)	38	25 (66%)	4 (10.5%)	9 (32.5%)
Third quarter	11	10	..	1	4	3	..	1	18	8	5	5
Fourth quarter	2	2	..	..	5	3	1	1	9	6	..	3
Total second 6 months	13	12	..	1	9	6	1	2	27	14	5	8
Total first year	94	69 (73.4%)	14 (14.8%)	11 (11.7%)	50	33 (66%)	8 (16%)	9 (18%)	65	39 (60%)	9 (13.8%)	17 (27%)
First half second year	6	5	1	..	8	6	1	1	14	9	1	4
Second half second year	5	5	..	..	4	3	1	..	9	7	..	2
Total second year	11	10	1	..	12	9	2	1	23	16	1	6
Total first 2 years	105	79	15	11	62	42	10	10	88	55	10	23
2-3 years	4	4	..	..	5	3	..	2	..	..	..	..
Total first 3 years	109	83 (76%)	15 (14%)	11 (10%)	67	45 (67.5%)	10 (18%)	12	88	55 (62.5%)	10 (11.3%)	23 (26.2%)

3. During the whole of the first year the percentages of normal and Babinski phenomena are again practically the same for the three different varieties of heads, although a Babinski phenomenon is somewhat less common among the brachycephalic group.

4. During the second year, the numbers are too few to make any comparison.

5. For the whole period of the first two years, we have the following figures:

Dolichocephaly: 105 cases. Out of these

79, or 75.2 per cent. had a normal reflex.
15, or 14.3 per cent. had a Babinski phenomenon.
11, or 10.5 per cent. had a variable reflex.

TABLE 9.—DOLICHO AND MESOCEPHALY; BRACHYCEPHALY

Age	Dolicho and Mesocephaly				Brachycephaly			
	No. of Cases	F	E	V	No. of Cases	F	E	V
First quarter	80	56	16	8	16	9	3	4
Second quarter	42	28	5	9	22	16	1	5
First 6 months	122	84 (69%)	21 (17%)	17 (14%)	38	25 (70%)	4 (10.5%)	9 (23.7%)
Third quarter	15	13	..	2	18	8	4	5
Fourth quarter	7	5	1	1	9	6	..	3
Second 6 months	22	18	1	3	27	14	5	8
Total first year	144	102 (70.8%)	22 (15.2%)	20 (14%)	65	39 (60%)	9 (30.8%)	17 (14%)
Second year	23	19	3	1	23	16	1	6
Total first 2 years	167	121 (71.2%)	25 (15%)	21 (12.5%)	88	55 (62.5%)	10 (11.3%)	23 (26.2%)
Third year	9	7	..	2	..	..	..	..
Total first 3 years	176	128 (72.7%)	25 (14.2%)	23 (13%)	88	55 (62.5%)	10 (11.3%)	23 (26.2%)

Mesocephaly: 62 cases. Out of these

42, or 67.7 per cent. had a normal reflex.
10, or 16.1 per cent. had a Babinski phenomenon.
10, or 16.1 per cent. had a variable reflex.

Brachycephaly: 88 cases. Out of these

55, or 62.5 per cent. had a normal reflex.
10, or 11.3 per cent. had a Babinski phenomenon.
23, or 26.2 per cent. had a variable reflex.

In other words, the incidence of a Babinski phenomenon is slightly less among the brachycephalic group than among the other two groups, suggesting that cerebral inhibition may be greater in that group of cases. The fact that no difference is shown during the first three months may possibly be accounted for on the supposition that the head has as yet not completely recovered from the effects of obstetric molding.

6. For all cases throughout the first three years the percentages are practically the same as for the first two years.[35]

Influence of the Nature of the Infant's Food on the Nature of the Plantar Reflex.—(Table 10) It has been alleged that the reason why human milk contains more lecithin and lactose than that of the cow is because more lecithin and lipin derivatives of lactose are required by the quickly growing infant's brain and nervous tissues with their myelin sheaths than is required by the more slowly growing nervous

TABLE 10.—INFLUENCE OF NATURE OF FOOD UPON THE NATURE OF THE PLANTAR REFLEX

Age	Breast				Bottle			
	No. of Cases	F	E	V	No. of Cases	F	E	V
First month	12	7 (58.3%)	3 (25%)	2 (16.7%)	9	4 (44.4%)	4 (44.4%)	1 (11.2%)
Second month	16	6	6	4	7	5	..	2
Third month	13	3	8	2	11	4	5	2
First quarter	41	16 (39%)	17 (41%)	8 (20%)	27	13 (48.1%)	9 (33.3%)	5 (19.9%)
Fourth month	8	5	2	1	6	4	2	..
Fifth month	1	..	..	1	2	..	1	1
Sixth month	2	1	..	1	3	1	..	2
Second quarter	11	6 (54.5%)	2 (18.2%)	3 (27.3%)	11	5 (45.5%)	3 (37.3%)	3 (27.2%)
Total first 6 months	52	32 (42.3%)	19 (36.5%)	11 (21.2%)	38	18 (46.3%)	12 (31.6%)	8 (21%)
Seventh month	5	4	..	1	2	..	..	2
Eighth month	3	2	..	1	1	1	..	..
Ninth month	2	..	2	..	..	..	..	..
Third quarter	10	6	2	2	3	3	..	..
Fourth quarter	3	2	..	1	9	6	1	2
Total second half year	13	8 (61.5%)	2 (15.4%)	3 (23.1%)	12	7 (58.3%)	1 (8.4%)	4 (33.2%)
Total first year	65	30 (46.1%)	21 (32.3%)	14 (21.6%)	50	25 (50%)	13 (26%)	12 (24%)
Second year	9	2	3	4	3	1	2	..
Total first 2 years	74	32 (43.2%)	24 (32.4%)	18 (24.4%)	53	26 (50%)	15 (28.3%)	12 (22.7%)

system of the calf.[32] If that is true, and if it is also true, as it practically certainly is, that to a certain extent the nature of the plantar reflex depends on the degree of myelination of the pyramidal tracts, then we would expect that in infants artifically fed there should be a larger percentage of dorsiflexor responses than in those fed on the breast. This is not found to be the case.

35. In view of the fact that owing to the development of the frontal sinuses and the occipital protuberance in later life the cephalic index slightly decreases with age, it is, perhaps, safer to classify both the dolichocephalic and mesocephalic types in our series together as dolichocephalic. This I did in Table 9. The results again show a smaller percentage of Babinski phenomena in the brachycephalic group.

During every age period (except during the first month) breast fed infants show a slightly larger percentage of Babinski phenomena than bottle fed infants, showing that breast feeding per se does certainly not tend to diminish the incidence of the Babinski phenomenon *after* the first month. It is not safe to generalize from the few cases during the first month, but as the percentages of Babinski phenomena during that period are 25 and 44 per cent., respectively, in breast fed and bottle fed babies, it would seem that breast feeding during the first few weeks has a salutary effect on the development of the nervous system.

If, further, we transfer from the thirty-eight bottle fed infants during the first half year (of which eighteen gave a flexor, twelve an extensor and eight a variable response), the fourteen infants (from the second month onward) which were breast fed for a variable period between two weeks and two months after birth (and of which seven gave a flexor, four an extensor and three a variable response), to the fifty-two that were entirely breast fed (up to the time of the examination) during the first half year, we obtain the figures shown in Table 11.

TABLE 11.—INFLUENCE OF NATURE OF FOOD ON PLANTAR REFLEX

	No. of Cases	F	E	V
Bottle fed first 6 months	38	18	12	8
Subtract partly breast fed	14	7	4	3
Entirely bottle fed	24	11 45.6%	8 33.3%	5 21%
Total entirely breast fed first 6 months	52	22	19	11
Add partly breast fed	14	7	4	3
Total breast fed (entirely or partly)	66	29 44%	23 34.8%	14 21.2%

In other words, we still arrive at the same result, that whatever influence breast feeding may possibly have on the development of the pyramidal tracts during the first month it has no effect whatever on the subsequent development of these tracts.

Influence of Body Temperature (Table 12).—A consideration of certain facts with regard to the circulation, respiration, and the superficial area of the skin in the infant leads one to expect that changes in the body temperature might have some influence on the nature of the plantar reflex in early life.

I have already shown that at birth the pyramidal tracts of the spinal cord are sufficiently well developed to conduct inhibitory impulses from the brain which would tend to produce a normal plantar reflex, and that the production of a Babinski phenomenon in early life is favored by insufficient cerebral control (? e. g. in dolichocephaly), as well as other factors, such as circulatory disturbances which might cause slight pressure on the incompletely developed tracts.

TABLE 12.—Influence of Body Temperature

Age	Subnormal				Normal				Pyrexia			
	No. of Cases	F	E	V	No. of Cases	F	E	V	No. of Cases	F	E	V
5 days	2	1	1	..	..	..	..	..	..	..	..	..
2 weeks	4	2	1	1	2	1	1	..	..	..	..	..
2–3 weeks	2	2	..	..	4	3	..	1	2	1	..	1
4 weeks	3	1	2	..	8	6	1	1	..	..	..	..
5 weeks	2	2	..	..	6	4	1	1	2	1	..	1
6 weeks	2	2	..	..	10	7	2	1	1	1	..	..
7 weeks	5	3	..	2	6	5	..	1	1	..	..	1
8 weeks	7	6	1	..	19	16	1	2	6	5	1	..
9 weeks	1	1	..	..	5	2	1	2	..	..	..	..
10 weeks	4	3	1	..	10	5	5	..	1	1	..	..
11 weeks	2	..	2	..	3	1	1	1	2	1	1	..
12 weeks	10	6	4	..	24	15	5	4	3	3	..	..
Total first 3 months	44	29 (66%)	12 (27%)	3 (7%)	97	65 (67%)	18 (18.5%)	14 (14.4%)	18	13 (72%)	2 (11%)	3 (17%)
Fourth month	9	5	1	3	19	12	5	2	8	5	1	2
Fifth month	4	2	1	1	8	5	..	3	5	5	..	..
Sixth month	3	3	..	..	13	11	..	2	5	3	..	2
Second quarter	16	10 (62.5%)	2 (12.5%)	4 (25%)	40	28 (70%)	5 (12.5%)	7 (17.5%)	18	13 (72.3%)	1 (5.5%)	4 (22.2%)
First half year	60	39 (65%)	14 (23.3%)	7 (11.7%)	137	93 (67.8%)	23 (16.8%)	21 (15.5%)	36	26 (72.2%)	3 (8.3%)	7 (19.5%)
Second half year	11	7 (63.3%)	2 (18.2%)	2 (18.2%)	27	19 (70%)	1 (4%)	7 (26%)	13	9 (70%)	2 (15%)	2 (15%)
Total first year	71	46 (64.8%)	16 (22.5%)	9 (12.7%)	164	112 (68.3%)	24 (14.6%)	28 (17.1%)	49	35 (71.4%)	5 (10.2%)	9 (18.4%)
Second year	1	1	..	..	33	23	5	5	16	10	5	1
Total first 2 years	72	47 (65.3%)	16 (22.2%)	9 (12.5%)	197	135 (68.5%)	29 (14.3%)	33 (16.7%)	65	45 (69%)	10 (15.5%)	10 (15.5%)

Now let us consider the following facts: (1) Czerny and Kleinschmidt [36] have recently shown radiographically that in severe disturbances of nutrition, which are generally accompanied by a subnormal temperature, and in which the abdominal muscles become relaxed, the heart becomes actually smaller in size on account of the interference with the movements of the diaphragm, which during inspiration helps to drive blood out of the abdomen into the thorax; (2) the relation between the surface and volume (or weight) of the body is greater in the infant than in the adult,[32] e. g. at birth the area of the cutaneous surface of the body per kilogram body weight is three times, and at one year twice that in the adult, and hence in cases of subnormal temperatures in which there is anemia of the skin there is a much greater congestion of the internal organs in the infant than there is in the adult.

Consequently, we would expect subnormal temperatures in infancy to bring about great venous congestion of all the internal organs, including the spinal cord, which would tend to produce a Babinski phenomenon. The opposite would be the case in conditions accompanied by pyrexia.

I have, therefore, classified my cases into three groups, viz. (1) those with subnormal temperatures (under 97 F.); (2) those with normal temperatures (between 97 and 99 F.), and (3) those with pyrexia (above 99 F.). The results are very striking (Table 11). They show that: At all periods from birth (0) to 3 months, from birth (0) to 6 months, and from birth (0) to 12 months, the incidence of cases of Babinski phenomena occurs in decreasing percentages in the subnormal, normal and supernormal temperature groups.

Influence of Certain Toxic Agents (see Protocols of Cases).—(1) Syphilis: Of six cases of congenital syphilis, during the first three months, five gave a normal adult type of response and one gave dorsiflexion on one side and plantar flexion on the other. The syphilitic virus, therefore, had no deleterious influence on the pyramidal tracts. Indeed, one would not, a priori, expect it to have. For syphilis among adults, while it may cause degeneration of the posterior columns (as in tabes), hardly ever causes degeneration of the pyramidal tracts, except secondarily to cortical degeneration (as in dementia paralytica). (2) Toxemia from the bowels: This does not seem to have any influence on the nature of the response. (3) Pertussis: It is believed by some that the attacks in whooping cough are due to toxic irritation of the vagus, but out of four cases of pertussis during the first two years, three gave a plantar flexor response and only one a dorsiflexion

36. Czerny and Kleinschmidt: Weiterer Beitrag zur kenntniss der Zirkulationsstörungen bei akuten Erunährungsstörungen der Säuglinge, Jahrb. f. Kinder. **84**:441, 1916.

of the toe. The number of cases is too few to generalize but it does not seem as if the pertussis toxin has any influence on the pyramidal tracts.

Other Factors.—(1) Rickets: has been alleged to retard the development of the pyramidal tracts. I do not find it to be the case. (2) Convulsions: Esmenard [37] finds that both during an epileptic fit in adults and soon after there is a Babinski phenomenon. In the case of infants suffering from convulsions, in the interval between fits there was a normal plantar reflex in all the four cases that came under my observation during the first two years of life, but in one case of convulsions and pertussis, in a girl, aged 4 years, there was a very marked Babinski phenomenon nine hours after a fit. (3) Sleep: most cases in which the reflex was examined during sleep were of the Babinski character.

DIFFUSION OF THE REFLEXOGENOUS ZONE

Bertolotti,[38] in 1904, and Laurent, in 1905, found that the area of skin, stimulation of which will produce a plantar reflex in normal infants, is not confined to the sole of the foot but is very diffuse. Thus in 10 per cent. of cases Bertolotti could elicit a downward movement of the toes on pinching the skin as high up as the abdomen, and in from 30 to 50 per cent. he elicited movement on pinching the skin in various parts of the leg and thigh (compare Oppenheim's, Gordon's and other modifications of the Babinski sign in adults). Laurent found a similarly great extension of the reflexogenous zone, but the abstract of his thesis (which is the only record of his results that has been accessible to me) does not state whether the response he obtained was a plantar flexion or dorsiflexion of the toes. Laurent further found that the extension of the zone tends to disappear after the age of 9 months. Yoshimura [17] confirms the extensive diffusion of the zone in infancy.

I have no statistics to offer on the matter, but I have examined a considerable number of cases and can fully confirm the findings of these observers with regard to the remarkable diffusion of the reflexogenous zone in early life. In quite a large proportion of cases—certainly more than the 10 per cent. found by Bertolotti—I have been able to obtain a plantar reflex on pinching the skin of the abdomen, even at the age of 1 year and 3 months.

In most cases the response was flexor in type (i. e., plantar flexion) but occasionally an extensor response was elicited, even when the

37. Esmenard, J.: Contribution a l'etude du phénomène des orteils dans l'epilepsie, These de Paris, 1902.

38. Bertolotti, M.: Etude sur la diffusion de la zone reflexogene chez les enfants, Rev. neurol. **12**:1160, 1904.

ordinary response was flexor. It is remarkable, however, that the response was almost in every case much more pronounced than when the stimulus was applied to the sole. After about eighteen months, the reflexogenous zone gradually shrinks. In two cases (Cases 759 and 766), in children between 3 and 4 years of age, in which the zone was localized to the sole of the foot, stimulation of the inner border produced plantar flexion of the big toe, while stimulation of the outer border evoked a dorsiflexion of the toe. This peculiarity was bilateral and constant in each case.

Another interesting fact is that although the reflexogenous zone is extensively diffused in a vertical direction, it is in the large majority of cases lateralized, i.e., pinching of the skin on one side will not, as a rule, produce a plantar response on the other side.

SUMMARY

I have examined the plantar reflex of about 500 subjects from birth up to the age of 7 years, the large majority being under 4 years. I have not recorded the exact number of cases at the various age periods in which no response could be elicited, because absence of a plantar reflex is not peculiar to infancy. In a good many cases in adults I have failed to obtain any plantar response. Moreover, absence of the response is entirely due to an affection or deficient development of the reflex arc and gives no information regarding the upper motor neuron with which alone I was concerned in this investigation. As, however, I was able to obtain a response in 426 cases, I conclude that the total percentage of nonrepsonses is about 15. In this respect, therefore, my figures are more in agreement with those of Engstler who, in a series of 1,000 cases found the reflex to be absent in 109 (11 per cent.), and of Finizio who among 500 new-born infants failed to elicit it in 5 per cent., than with the 35 per cent. of absent reflexes given by Morse, and 40 per cent. given by Schüler.

As regards the nature of the response, my findings agree very closely with those of Finizio. For while all other observers find the prevailing response to be of the Babinski type, both Finizio and myself find the proportion between plantar flexion and dorsiflexion to be approximately 4:1 (Table 1). I cannot, however, confirm Finizio's statement with respect to the influence of forceps delivery on the nature of the response, for while Finizio finds that in difficult forceps cases there is dorsiflexion of the toe, of the two new-born forceps cases in my series, one of which (Case 602) was a particularly difficult one and gave rise to a good deal of trauma, both gave a normal response (i. e., plantar flexion). But my numbers are too few to draw any conclusions. I give my results as I have found them.

As regards the age at which the plantar reflex definitely becomes plantar flexor in type, I cannot agree with the conclusions of some observers that it depends on the age at which the child begins to walk, since not only do I find plantar flexion to be the prevailing response before the walking age, but I have obtained it in a few cases either unilaterally or bilaterally, long after the child had begun to walk. Moreover, in the same infant you might at different times elicit either plantar flexion or dorsiflexion.

As regards the correlation between the degree of development of the pyramidal tracts and the nature of the response, and the influence of the various developmental and environmental factors on it, see pp. 3 and 4, as well as under "conclusions."

CONCLUSIONS

1. The prevailing plantar response in early life is plantar flexion of the big toe, although when a dorsiflexion of the toe occurs it has not the same significance as a similar response in the adult.

2. The pyramidal tracts are sufficiently developed at birth (in cases of birth at full term) to give a normal adult type of plantar reflex even intra-uterinally, but owing to easily aroused circulatory disturbances in early life, the consequent changes in the circulation in the region of the cord are sufficient to compress the incompletely myelinated pyramidal tracts to evoke a Babinski phenomenon either unilaterally or bilaterally.

3. In premature infants the response is nearly always of the Babinski type, up to 5 or 6 weeks postnatal life, because of the almost total absence of myelination of the pyramidal tracts.

4. Malnutrition, as judged by defective weight and length, is not in itself sufficient to give a Babinski sign, but inasmuch as prematurity and defective weight and length go hand in hand up to about 5 or 6 weeks, the Babinski phenomenon seen in badly developed infants up to that age is due to the prematurity rather than to the malnutrition.

5. Bilateral plantar flexion is at all ages as common in girls as in boys, but bilateral dorsiflexion is at all age periods in infancy more common in girls than in boys.

6. Breast feeding during the first few weeks of life probably tends to diminish the incidence of a bilateral Babinski phenomenon; this may be so owing to the greater percentage of lecithin and lactose in human milk which helps the more rapid myelination of the pyramidal tracts. After the first month or so breast feeding has no advantage in this respect over bottle feeding.

7. Toxic influences, either from the bowel or from other causes, do not affect the conductivity of impulses along the fibers of the pyramidal tracts.

8. Bilateral dorsiflexion of the toes is commoner in cases with a subnormal temperature because, probably, in such cases there is a greater congestion of the spinal cord as the result of: (a) the pallor of the skin, whereby a relatively much larger quantity of blood goes to the internal organs (including the spinal cord) than in adults; (b) the interference with the action of the diaphragm and consequently of the pumping action of the heart in cases of severe disturbances of nutrition—which are frequently the accompaniments of subnormal temperatures.

9. Bilateral dorsiflexion is slightly more common in dolichocephalic than in brachycephalic infants, possibly because inhibitory control is less powerful in the former than in the latter.

10. Rickets does not favor the occurrence of a Babinski phenomenon.

11. The age at which the Babinski sign vanishes has no relation to the age at which the child begins to walk. In the majority of cases of very young infants who cannot even sit up the Babinski sign is absent, and in the cases of a large number of children who can walk and are "strong on their legs" (in some even during the third and fourth years) the Babinski sign is present.

12. As the peripheral nerves are imperfectly myelinated at birth [32] a possible explanation of a Babinski phenomenon in certain infants is more imperfect development of the lower motor neuron supplying the flexors of the toes but better development of the neuron supplying the extensors. In such cases, of course, extension is the only possible movement.

13. The inconstant nature of the response in certain infants in whom at the same examination one may obtain on one stimulation a plantar flexion and on another stimulation of either the same or a different cutaneous area a dorsiflexion may be due to the easy fatigability, as well as easy recovery from fatigue of muscle in early life, so that after a certain response has been obtained the muscles producing that response can no longer contract as easily as the opposing groups of muscles and the response is, therefore, produced by the less fatigued group of muscles.

14. The reflexogenous zone is very diffuse in early infancy, and sometimes one is able to elicit a plantar reflex (either flexor or extensor) by stimulating a cutaneous area other than the sole, when stimulation of the sole fails to evoke a response.

PROTOCOLS OF CASES

Age 0 (At Birth) Normal Weight, 7 Pounds

Case No.	Sex	Weight, Lbs.	Direction of Toe	Remarks
615	M	8	↑↓ each	Normal birth (Case 616, age 0-24 hours)
715	F	9¼	F	Forceps
716	F	3¾	E	6½ months' fetus
717	F	About normal	F	Reflex tried intra-uterinally and postnatally; transverse presentation; internal version under chloroform; baby born with two lower incisors
718	M	6½	F	Placenta praevia

Forceps, intra-uterine manipulation and placenta praevia did not produce a Babinski phenomenon, but prematurity did.

Age, 0-24 Hours

Case No.	Sex	Weight, Lbs.	Direction of Toe	Remarks
616	M	8	E (at 1 hr.)	Age = 1 hour; same case at birth was V (Case 615)
614	M	8	F (at 14 hrs.)	This infant died suddenly at the age of 25 hours (see notes, postmortem report and microscopic report of cord sections, p. 9)
605	F	6	F (at 22 hrs.)	

Age, 1-2 Days

614	M	8	V	Age = 28 hours; see Case 614 under 0-24 hours

Age, 3 Days

602	F	10	F	Difficult forceps labor; hematoma of neck and contusion of face; convulsions on second day; mother suffered from dropsy for a few weeks before confinement

Forceps and trauma of birth, as well as convulsions on second day did not produce a Babinski sign.

Age, 5 Days *

Case No.	Sex	Weight, Lbs.	Length, In.	Temperature	Cephalic Index	Direction of Toe	Feeding
729	M	3.15	16½	96.2	87	F	Bottle
730	M	3.11	17	96.2	81	E	Bottle

* These two babies were twins. They were prematurely born at 7½ months. Mother suffered from puerperal sepsis. In spite of the identical sex and prenatal as well as neonatal environments, etc., the plantar reflex was flexor in one and extensor in the other.

Age, 1-2 Weeks

Case No.	Sex	Weight, Lbs.	Direction of Toe	Remarks
606	F	8	F	Age, 11 days
607	F	4	E	Age, 12 days

Explanation of abbreviations and symbols: Under sex, M = male: F = female. Under direction of toe, F = plantar flexion (flexion); E = dorsiflexion (extension); V = variable (different either on the two sides or on the same side at different times);

↑ = dorsiflexion; ↓ = plantar flexion; o = no reflex.

$\begin{matrix}\uparrow\\\downarrow\end{matrix}\begin{matrix}R\\L\end{matrix}$ = dorsiflexion on right side, plantar flexion on left side.

$\begin{matrix}\uparrow\\\downarrow\end{matrix}\begin{matrix}L\\R\end{matrix}$ = dorsiflexion on left side, plantar flexion on right side.

$\begin{matrix}\uparrow\\o\end{matrix}\begin{matrix}L\\R\end{matrix}$ = absent reflex on right side, dorsiflexion on left side.

$\begin{matrix}o\\\uparrow\end{matrix}\begin{matrix}L\\R\end{matrix}$ = absent reflex on left side, dorsiflexion on right side.

$\begin{matrix}o\\\downarrow\end{matrix}\begin{matrix}R\\L\end{matrix}$ = absent reflex on right side, plantar flexion on left side.

$\begin{matrix}o\\\downarrow\end{matrix}\begin{matrix}L\\R\end{matrix}$ = absent reflex on left side, plantar flexion on right side.

V $\begin{matrix}\uparrow\\\downarrow\end{matrix}$ each = variable on each side.

PROTOCOLS OF CASES

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
Age, 2 Weeks; Normal Weight, 7½ Pounds, and Length, 20 Inches									
4	M	Icterus, neonatorum	6.13	18½	96.4	72.7	?	E	
166	F	Uric acid calculus	6.0	19¾	96	72.2	?	F	
220	F	Tongue tie	6.15	21	98	82.6	?	F	
505	F	Wasting	2.12	17	97	75	?	↑ R ↓ L	Prematurely born at 8 months
683	F	Screaming	7.2	21	96.4	....	Breast	F	
751	M	.....	2.4	....	98	80	Bottle	E	7 months' fetus; weight at birth, 3½ pounds
761a	F	Crying	8	....	....	....	Bottle	F	
35	F	Spina bifida	4.14	16	....	72	Breast	E	Eighth infant, full term
Age, 2-3 Weeks									
285	M	Flatulence	6.15	19	97	75	Breast	F	Age, 16 days
608	M	Nil	6.14	....	98.4	....	Breast	F	Forceps delivery; age, 17 days
609	M	Nil	6.8	....	....	....	Breast	E	Age, 19 days
789	M	Crying	8	....	98.4	....	Bottle	F	Age, 18 days
Age, 3 Weeks									
213	M	Ophthalmia neonatorum	5.1	17¾	97	88	Bottle	F	Mother consumptive
244	M	Diarrhea	Very tiny infant				Bottle	E	Very feeble, puny infant; collapsed; brandy and oxygen given; premature
276	M	"Always hungry"	8	21¼	98.4	80	Breast	F	
655	M	Flatulence	7.12	22	98	....	Breast	V ↑ L ↓ R	
658	M	Swollen feet	9.3	22½	99	68	Breast	V ↑ R L	? Idiopathic dropsy
481	M	Vomiting	7.14	23¾	100.2	80	Bottle	F	
Age, 4 Weeks; Normal Weight, 7¾ Pounds; Length, 20½ Inches									
128	M	Wasting	5.3	26	98.4	85	Bottle	V ↑ R ↓ L	This was a baby with congenital absence of fibula and tarsal bones; curvature of tibia; see notes roentgen ray report, postmortem report and notes on sections of card, p. 9
258	?	Wasting	8	19	96.2	76	?	E	7 months' fetus
299	M	Vomiting	7.7	21	97.8	84	?	F	
335	M	Erythema	10.8	22	98.4	77	?	F	
329	M	Right inguinal hernia	4.9	17	96	75	Bottle	E	7½ months' fetus
375	F	Flatulence	6.8	19	98.2	75	?	F	
423	M	Wasting	4.6	16¼	96	79	?	F	Weight at birth, 5 pounds; ? full term
453	M	Phimosis	9.8	21¾	98.2	70	?	F	
470	M	Flatulence	10.8	24½	98	77	?	F	
581	F	Screaming	8.12	21	98	84	Breast	E	
610	M	Nil	7.14	....	....	....	Breast	F	
711	M	Rash on head	8.3	25	98.4	....	Breast	F	
Age, 5 Weeks									
132	F	Wasting, congenital syphilis	6.1	18¼	98.2	80	.....	F	
148	F	Wasting	7.13	20½	97.6	93	.....	F	
167	M	Diarrhea and vomiting, wasting	7.2	20½	99.6	70	.....	F	
183	F	Rash, (?) toxic	8.11	20½	98.4	79	.....	E	Infant asleep
305	F	Fretful	6.2	18¾	97	75	.....	F	
307	M	Vomiting	6.2	18½	96	75	.....	F	
449	M	Sore buttocks	9.6	19¾	98.8	76	.....	F	
458	M	Snuffles and nasal obstruction	11.3	22½	98.4	72.7	.....	F	
608	F	Vomiting	7.7	18½	....	....	Bottle	F	Weight at birth, 4 pounds; premature; breast fed only one day

PROTOCOLS OF CASES

Age, 5 Weeks—(Continued)

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
611	M	.....	10	....	....	....	.....	F	
631	M	Constipation	8.12	23½	99	76	Bottle	V	Breast fed first 2 weeks
703	M	Constipation	9.5	22	97.4	....	Breast	V	
704	M	Screaming	11.8	22	97	....	Breast	F	
739	F	Wasting	5.9	18½	96.2	77.7	Breast	E	8 months' fetus weaned at one month

Age, 6 Weeks

12	F	Diarrhea and	8.10	21½	98.6	76	.....	F	
275	F	Nil	8.8	21	97.4	80	.....	F	
279	M	Wasting	6.15	21	98	80	.....	V ↑ L ↓ R	
282	F	Bronchitis	12.12	23¾	97.4	81.8	.....	F	
292	F	Flatulence	9.8	20¾	99	77	.....	F	
322	M	Constipation	8.2	22	97	71.4	.....	F	One of twins
341	F	Bronchitis	6.11	19½	96	76	.....	F	
456	M	Screaming	9.9	20½	97.2	77	.....	F	
461	F	Umbilical hernia	7.4	20	97	80	Bottle	F	Congenital syphilis; on breast 2 weeks
463	M	Systolic bruit all over heart	7.9	20½	98.4	80	Breast	F	(?) patent foramen ovale
582	F	Vomiting 10 days	8.15	21¼	98	84	Breast	E	
640a	F	Nil	....	....	....	....	Bottle	F	Same case as 602 (see under 3 days)
712	M	Right inguinal hernia	9.11	23½	98.4	72.7	Breast	F	
713	F	Flatulence	11.4	23½	98.2	81	Breast	E	
666	F	Wasting and vomiting	3.11	17	98.8	77.7	Bottle	F	7 months' fetus
678	?	.....	....	....	....	....	.....	F	See also same case under 1 yr. (681)
681	?	.....	....	....	....	80	.....	F	See also under 1 year = V
752	M	Nil	9	21½	....	....	Breast	F	

Age, 7 Weeks

242	M	Wasting	8.5	22	96	76	.....	F	
345	M	Rash, (?) toxic	10.12	21	97	77	.....	V ↑ L ↓ R	
375	F	Flatulence	6.8	19	98.2	75	Bottle	F	Breast 3 weeks
425	M	Screaming	7.6	20	97	82.6	.....	F	
445	F	Vomiting	7.5	22	98.4	84	.....	F	
446	F	Constipation	6.12	19¾	96.6	80	.....	F	
482	F	Thrush	12	21	98	77	.....	F	
653	M	Glands in neck (?) C. S.	10.15	23¼	99.2	....	Breast	V ↑ R ↓ L	
662	M	Malnutrition	7.4	23½	98	77	Breast	V ↑ L ↓ R	
706	F	Wasting	5.14	19½	96	....	Bottle	V ↑ L ↓ R	
707	M	Umbilical hernia	9.4	21½	98.8	....	Breast	F	
733	M	Screaming	10.13	22½	98.2	82	Breast	F	
762	F	Screaming		Well developed			Breast	F	

Age, 2 Months (8 weeks); Normal weight, 9½ Pounds; Length, 21 Inches

1	M	Wasting and diarrhea	9.6	21½	98.4	73	.....	F	
13	F	Vomiting	11.2	20¾	99.8	96	Bottle	F	Breast 2 weeks
109	M	Wasting and diarrhea	9.8	21½	98.4	73	.....	F	Twelfth infant
129	M	Bronchopneumonia	11.10	23	102	86	.....	F	
142	M	Wasting and vomiting	7.7	21	97	79	.....	F	
163	F	Wasting and diarrhea	7.0	19½	98	80	.....	F	
170	F	Flatulence	9.6	23	99	79	.....	F	
172	M	(?) C. S.	8.5	22	97.6	80	.....	F	
334	F	Vomiting	9.10	21½	97.6	77	.....	F	
174	M	Fretful and wasting	7.4	21½	97	76	.....	F	
194	M	Wasting	7.10	21¼	98	84	Breast	V ↑ ↓	each
223	F	Adenoids	9.4	21¾	98	81	.....	F	

PROTOCOLS OF CASES

Age, 2 Months (8 weeks); Normal Weight, 9½ Pounds; Length, 21 Inches—(Continued)

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
339	M	Icterus neonatorum	7.5	20	97.2	76	.....	F	7 months' fetus
289	M	.....	16	....	97.4	84	.....	V ↑ L ↓ R	
388	F	Wasting	6	19½	96	76	.....	F	
417	M	Bronchitis	9.4	22	97	80	.....	F	Father and paternal aunt died of consumption, maternal uncle suffering from consumption
428	F	Wasting and vomiting	6.2	20	99.6	79	.....	F	
430	F	Constipation	10.6	22½	97.6	76	.....	F	
436	F	Rash	10.12	21¾	97	73	.....	F	
447	F	Flatulence	9.8	20½	98	81	.....	F	
457	F	(?) C. S.	5.4	20	97.4	70	.....	F	
460	F	Not progressing	7.7	21	97	80	.....	F	
459	F	Constipation	13.1	22½	99.2	81	.....	F	
469	M	Wasting	7.12	19¼	98	76	.....	F	
487	F	Wasting	4.7	17	96.6	79	.....	F	} Premature twins
488	M	Cough	5.5	18	98	75	.....	F	} at 8 months
626	F	Vomiting and crying	7.1	20	98	....	.....	F	
638	F	Snuffles	8.12	20½	97	....	Breast	E	
639	M	Constipation	7.15	21	97.2	....	Breast	E	
677	F	Umbilical hernia	9.13	24	97.4	....	Breast	F	
708	M	Umbilical hernia and double inguinal hernia	10.8	21½	98	....	Bottle	F	Breast fed 3 weeks
712	M	Inguinal hernia	9.11	23½	98.4	73	.....	F	
740	F	Wasting	7.15	22¼	99	81	Breast	E	
743	M	Wasting and vomiting	8.3	21	98.2	82	Bottle	F	Breast fed 2 weeks
					Age, 9 Weeks				
432	?	Wasting, flatulence	7.1	21½	97.2	72.7	.....	V ↑ L ↓ R	Same case as 432a; under 6 mos. = F
318	M	Phimosis	8.13	21½	97.8	77	.....	F	
673	F	Wasting	6.6	19	98	75	.....	F	
593	M	Inguinal hernia	9.0	20	98.4	81	.....	V ↑ L ↓ R	8 months' fetus
477	F	Screaming (?) C. S.	6.10	20½	97	76.2	.....	F	One of twins
737	F	Wasting and vomiting	8.0	22	97.2	76.2	Breast	E	
					Age, 10 Weeks				
208	F	(?) Pertussis and wasting	9.14	23½	97.4	80	.....	E	
226	F	Bronchitis	8.4	20½	98	90	.....	F	
312	F	Wasting	7.3	20	96.4	71	.....	F	
431	F	Cough	8.6	20	97	76.2	.....	E	
325	F	Vomiting	10.15	22	98.4	72.7	.....	F	
452	M	Rectal prolapse	11.1	20½	99.6	72.7	.....	F	
600	M	Wasting	4.9	17	97	79	.....	F	} Twins; premature;
601	M	Wasting	5.10	18½	97.8	95	.....	F	} fifth pregnancy
629	M	Constipation	10.8	23½	98.4	82.6	Breast	F	
643	M	Wasting	8	20	96.6	79	Bottle	E	Breast 2 weeks
644	F	.....	11.7	23	98	76	Breast	E	
659	M	Vomiting	11.10	23¾	99	....	Breast	V ↑ L ↓ R	
661	M	Malnutrition	7.8	22½	97.6	....	Breast	F	
684	M	Wasting	6.2	20½	98	79	Breast	E	
637	F	Wasting	5.13	18¾	97	....	Bottle	F	
744	M	Screaming	8.2	24½	98	85	Bottle	E	Breast 2 weeks
761	F	Wasting	7.12	....	....	....	Bottle	F	
					Age, 11 Weeks				
151	M	Wasting	7.4	20½	99	72.7	Breast	F	
311	F	Wasting	12.8	23	99	76	.....	E	
455	M	Wasting	6.15	18¾	98	76	.....	F	Father had splenectomy 8 mos. previously; paternal uncle had pernicious anemia
633	M	Constipation	11.2	23¾	97	....	Breast	E	
650	M	Wasting	6.5	20	97.2	....	Bottle	V ↑ R ↓ L	} Twins; premature
651	M	Wasting	6.11	20	97.6	....	Bottle	E	}
768	M	Wasting	8	19½	96	70	Breast	E	

PROTOCOLS OF CASES

Age, 3 Months; Normal Weight, 11 Pounds; Length, 22 Inches

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
8	M	(?) Toxic poisoning	12.5	25	98.4	78	.....	F	
14	M	Wasting	7.10	21½	97	80	.....	E	
24	F	Anemia and wasting	6.15	20½	97.4	81	.....	F	
26	M	Wasting	8.15	21	98.6	76.2	.....	F	8 months' fetus
57	F	Sore buttocks	10.12	22¼	96	86	.....	E	
68	M	Flatulence	8.13	20	98.4	86	.....	F	
93	F	Constipation	11.7	23½	97	77	.....	F	8 months' fetus
98	M	Losing weight	8.15	22¼	98	82	.....	F	Thirteenth infant: six died of wasting, one died of meningitis
112	F	Anemia	6.15	20½	97	81	.....	F	
119	M	Cough	8.13	22	97	76	.....	F	
136	M	Wasting	7.4	22	97	73	.....	F	
144	M	Rash (?) toxic	12.6	24	98.2	74	.....	V ↑ L ↓ R	
158	M	Wasting	9.4	23¾	103	71	.....	F	
165	M	Wasting	8.7	22½	98.2	81	.....	F	
168	M	Vomiting and screaming	11.5	23	97.2	81	.....	V	each
178	M	Diarrhea and vomiting	9.13	22½	96.6	74	.....	V ↑ L ↓ R	
186	M	Skin eruption	9.5	22	98.8	86	.....	F	
204	F	Erythema	10.11	21½	97	77	.....	F	
256	M	Constipation, sleeplessness	8.13	21¾	97.6	71	.....	E	
266	M	Wasting	11.1	23	98.4	74	.....	F	
304	M	Constipation	11.14	21¾	98	86	.....	F	
319	M	Diarrhea and vomiting	12.10	25	97.2	77	.....	F	
401	M	Cough	11.4	21½	100.4	70	.....	F	
419	F	Cough	15.4	21½	98	73	.....	F	
437	M	Wasting	8.4	20½	98.4	81	.....	E	
440	F	Wasting	8.13	20½	97	73	.....	F	
484	F	Wasting	6.0	19	98	70	.....	F	8 months' fetus
493	M	Wasting	6.10	19¼	96.2	76	.....	E	
545	M	Flatulence	8.4	21	97.2	76	Breast	F	
598	F	Malnutrition	7.7	19½	97.2	76	.....	V ↑ L ↓ R	
619	M	Screaming	....	....	....	....	Bottle	E	"Glaxo" baby
620	F	Nil	9.12	22½	97.4	86	Breast	E	
635	F	Cough	11.7	24	98.8	77	Breast	E	
640	M	Cough	.....	....	....	....	.....	E	Congenital hydrocephalus
664	F	Wasting	7.4	22¾	98.8	81	Bottle	F	
676	M	Wasting, C. S.	5.5	18¼	96.6	....	Bottle	F	Premature (8 mos.); breast fed 2 weeks
700	F	Rash on thigh	13.7	24½	99.2	....	Breast	E	
652	F	Supernumerary thumb	12.2	22¾	98.2	....	Breast	E	
728	F	Umbilical hernia, wasting	7.10	24	96	84	Bottle	V ↑ R ↓ L	
742	M	Wasting	6.15	23	98.4	85	Breast	V ↑ L ↓ R	
333	M	Wasting	9.10	21½	100.4	70	.....	F	
670	M	Wasting	7.2	23	97	76	Bottle	E	

Age, 14 Weeks

130	M	Wasting	7.9	20¼	97.4	76	.....	F	
274	M	Diarrhea and vomiting	14.3	25	98	90	.....	F	
421	M	Wasting	8.13	21½	97	78	Breast	F	
590	M	Umbilical hernia	9.11	22	98.4	76	Bottle	F	
654	F	Screaming	11.13	24	99.6	....	Breast	V ↑ L ↓ R	
667	M	Wasting	11.1	23	98.4	74	Breast	F	
685	F	Constipation	12.2	26	99.2	....	Breast	F	
624	M	Wasting	7.10	19	96	....	.....	V ↑ L ↓ R	

Age, 4 Months; Normal Weight, 12½ Pounds; Length, 23 Inches

5	F	Wasting and vomiting	8.9	19	97.2	76	.....	V ↑ L ↓ R	
11	F	Diarrhea and vomiting	12.8	24	96.6	79	.....	F	
90	M	Wasting and vomiting	10.4	22½	97	79	.....	F	

PROTOCOLS OF CASES

Age, 4 Months– Normal Weight, 12½ Pounds; Length, 23 Inches—(Continued)

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
106	F	Not gaining	8.9	19½	98.2	78	.....	V ↑ L ↓ R	
140	M	Left apical pneumonia with convulsions	9.5	21	103	81	.....	V ↑ R ↓ L	
160	M	Bronchitis	16.13	25½	96.6	82	.....	F	
164	M	Diarrhea and vomiting, wasting	11.15	24½	96	85	.....	V ↑ R ↓ L	
173	F	Malnutrition	8.14	....	98.6	80	.....	F	
175	F	Intestinal toxemia	9.2	21½	98.4	81	.....	E	8 months' fetus
254	M	Wasting and diarrhea	10.9	22	97.4	86	.....	F	
287	M	Ectopic vesicae	14.8	23	100	79	.....	F	
320	M	Seborrhea capitis, (?) imbeciility	10.8	24½	98	73	.....	F	
323	M	Malnutrition	7.1	....	98.2	73	.....	F	Premature
370	M	Wasting	8.12	24	101.8	83	.....	F	
408	M	Eczema; right inguinal hernia	13.6	23¾	97	79	.....	V ↑ R ↓ L	
473	F	Umbilical hernia, bronchitis	10.3	20½	99	76	.....	F	
604	F	Pertussis		Well developed			Breast	F	
622	M	Vomiting	.....	....	....	....	.....	E	
672	F	.....	12.10	24½	98.6	81	Breast	E	
674	M	Umbilical	10.7	22	98	....	Bottle	E	Twins, each breast-fed 1 month
675	F	hernia	12.2	23¾	98.2	....	Bottle	F	
684	M	Wasting	10.8	23½	99.2	....	.....	E	8½ months' fetus
701	M	Wasting	10.14	23	97	....	Bottle	F	Breast fed 2 weeks
422	M	Right inguinal hernia	9.9	21	97.8	73	.....	F	
599	F	Wasting	8.11	20	97.4	77	.....	E	
719	F	Slight hemahypertrophy of right side of face	16.8	26	98.4	....	Bottle	F	
265	M	.....	11.7	22	98	83	Breast	F	
306	M	Diarrhea	11.4	23½	99	79	.....	F	
741	F	Constipation	7.13	23½	98	76	Breast	E	
748	F	Screaming	10.4	25¼	97	90	Bottle	E	Breast fed 3 weeks

Age, 5 Months; Normal Weight, 14 Pounds; Length, 23½ Inches

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
27	F	(?) Pertussis	11.6	23½	99	70	.....	F	
33	M	Wasting	9.9	22¼	97.2	81	.....	V ↑ L ↓ R	
84	F	Wasting and bronchitis	11.2	26	97	75	.....	F	
105	F	Bronchitis	11.6	23½	99	70	.....	F	3 weeks premature
146	M	Diarrhea and vomiting, wasting	12	24	97.6	82	.....	F	
155	?	Diarrhea	15.6	27	98	86	.....	F	
162	M	Wasting	8.9	21½	96	85	.....	V	each
235	M	Wasting, pulmonary tuberculosis	8.0	25	97	74	.....	F	
283	F	Restlessness at night	13	24¾	99	83	.....	F	
324	F	Vomiting	7.4	20	96	71	.....	E	
412	F	Slight mongolism	11.2	21½	99	85	.....	F	
315	M	Bronchitis		Well developed			.....	F	
427	M	(?) Defective sight	15.4	27½	98	79	.....	F	
441	F	Digestive trouble	9.3	19¾	98	77	.....	F	
450	F	Wasting	13	23¼	98	76	.....	F	
458	M	Snuffles	11.3	22½	98.4	73	.....	F	
642	M	Cough	15.11	26	98	79	Breast	V ↑ L ↓ R	
732	F	Wasting	9.13	24¼	98.2	82	Bottle	V ↑ R ↓ L	Breast fed 2 mos.
763	F	Nil	17.4	....	....	....	Bottle	E	Same case as 719 under 4 mos. = F

Age, 6 Months: Normal Weight, 15 Pounds; Length, 24 Inches

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
2	F	Bronchitis	6.15	25	97	78	.....	F	
15	M	Diarrhea and vomiting, internal strabismus (due to error of refraction)	9.2	22¾	97.6	83	.....	F	

PROTOCOLS OF CASES

Age, 6 Months; Normal Weight, 15 Pounds; Length, 24 Inches—(Continued)

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
108	F	Bronchitis	16.5	25	97	74	.....	F	
153	M	Wasting, diarrhea and vomiting	10.13	23	97.4	81	.....	F	
176	M	Diarrhea and vomiting	11.12	24	98.4	85	.....	F	Twins, 6 weeks premature
177	M		8	21	98.6	77	.....	F	
201	M	Diarrhea	16.2	21½	99	87	.....	F	
360	M	Vomiting	15.11	27	98.8	80	.....	F	
389	F	Flatulence and cough	16.1	23½	97.8	80	.....	F	
390	M	Restlessness	17.12	25	99	80	.....	V ↑ L ↓ R	
400	M	Diarrhea	14.6	24	99	81	.....	F	7 weeks premature
442	M	Diarrhea and vomiting	14.11	25½	99.2	78	.....	F	
426	M	Diarrhea and vomiting	16.13	26	98.6	83	.....	F	
485	F	Rectal prolapse	.....	....	....	....	.....	F	
397a	F	.....	.....	...	....	.. .	.....	F	
432a	?	.....	.....	....	....	....	.....	F	Same case as 432 under 9 wks. = V
141	M	(?) Meningitis; fontanel bulging; (?) hydrocephalus	12.1	25	97.6	88	.....	F	
634	M	Wasting	7.6	23½	97.4	....	Breast	F	
665	M	Wasting	14.8	24	99	73	Breast	V ↑ L ↓ R	
594	M	.....	16.0	26	98.8	88	.....	F	
657	F	Diarrhea and vomiting	11.7	22½	97.8	82	Breast	V ↑ L ↓ R	
731	F	Wasting	7.4	22	98	82	Bottle	V ↑ R ↓ L	
735	M	Screaming	9.3	21½	98	85	Bottle	F	

Age, 7 months; Normal Weight, 16 Pounds; Length, 24½ Inches

82	M	Not thriving	13.8	23	99.6	83	.... ..	E	6 weeks premature, 4 lbs. at birth
95	M	Impetigo	15.2	25¼	97	87	.....	F	
161	F	Wasting	12.10	24	97.4	77	.....	F	
230	F	Rickets	12.5	24¼	98	78	.....	F	
281a	F	Well developed	.....	....	....	....	.....	F	
338	M	Rickets	18.9	28	98	80	.....	F	
357	M	Convulsions	15.14	28	98.2	83	.....	F	
586	M	Diarrhea and vomiting	15.13	26	98.4	75	Breast	F	8 months' fetus
591	F	Malnutrition	7.3	18¾	99	73	Breast	F	
596	M	.....	19.12	25½	99.2	75	.....	F	
623	M	Convulsions	9.5	....	97.2	....	.....	F	
656	M	Seborrhea capitis	17.11	26	98	....	Breast	F	Age, 7½ months
738	M	Wasting and vomiting	10.5	22½	97.8	77	Bottle	V ↑ R ↓ L	6½ months' fetus; breast fed 6 wks.
745	F	Wasting and vomiting	8.4	22½	96	83	Breast	V ↑ L ↓ R	During sleep
746	M	Wasting and vomiting	9.6	21¾	97.4	74	Breast	F	Twins
749	M	Diarrhea	9.3	20½	97.4	87	Bottle	V ↑ R ↓ L	

Age, 8 Months; Normal Weight, 17 Pounds; Length, 25 Inches

7	M	Diarrhea and vomiting	13.7	26¾	99	79	.....	F	
139	F	Wasting	8.3	22	98.4	83	.....	F	
149	M	Diarrhea and vomiting	8.12	23	97	82	.....	V ↑ R ↓ L	
225	F	Diarrhea	16.4	25½	99.2	85	.....	V ↑	each
234	F	Injury to leg; no fracture	20.2	28½	99	90	.....	F	
255	M	Mongolism; slight macroglossia; nystagmus; adenoids	9.7	26¾	98	83	.....	V	
406	M	Cough	17.5	28	99.6	80	.....	V ↑ L ↓ R	
451	F	Cough	16.12	27	99.4	76	.....	F	
485	M	Diarrhea	18.10	28½	100.6	79	Bottle	F	7 months' fetus; family history of tuberculosis; breast fed 2 wks.
413	F	.....	.....	....	....	86	.....	E	
660	F	Malnutrition	11.4	24	98	....	Breast	V ↑ L ↓ R	Age, 8½ months
632	M	Wasting	11.0	24	98	90	Breast	F	8 months' fetus
747	F	Bronchitis	20.3	31	98.6	73	Breast	F	

PROTOCOLS OF CASES

Age, 9 Months; Normal Weight, 18 Pounds; Length, 25½ Inches

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
185	M	Diarrhea	20.4	28½	97	84.6	.....	E	
206	M	Restlessness	21.4	32½	101.4	87	.....	F	
211	M	Ileocolitis	11.4	20¾	100.2	85.7	.....	F	
259	M	Cough	13.7	24½	99.2	78	.....	F	
294	M	Earache	16.3	29	97.2	80	.....	F	
310	F	Wasting	11.2	27	97	82.6	.....	F	
627	M	Malnutrition	11.9	23½	98.4	91	Breast	E	8 months' twins; age 9½ months
628	M		11.13	26	99	82.6	Breast	E	
433	M	Diarrhea and vomiting	10.6	22	97	73.7	.....	F	7 months' fetus

Age, 10 Months; Normal Weight, 19 Pounds; Length, 26 Inches

6	F	Bronchopneumonia	15.13	27	101.2	79	.....	F	
291	F	Diarrhea	17.11	27	97	85	.....	F	8 months' fetus
309	F	Rickets	12.10	25½	98.4	83	Bottle	F	
630	M	Cough	11.2	23¾	98.6	82	Bottle	F	
709	M	Diarrhea and vomiting	14	25	98.2	....	Bottle	F	
436	F	.....	.....	....	....	80	.....	F	
314	F	Diarrhea	17.7	27	98	74	.....	F	8 months' fetus; one of twins
476	F	Ileocolitis	10.14	....	98	82	.....	F	

Age, 11 Months; Normal Weight, 20 Pounds; Length, 26½ Inches

22	M	Rickets	11.7	24¾	....	79	.....	F	
138	F	Convulsions	22.7	29½	96.4	83	.....	F	Strong family history of tuberculosis
157	M	Bronchopneumonia; (?) tuberculosis	15.2	27	99.2	79	Bottle	F	Breast 3 months
263	F	Otorrhea and eczema	21.10	29½	97.2	83	.....	V ↑ R ↓ L	
649	M	Diarrhea and vomiting	20.3	29	97.4	....	Breast	V ↑ R ↓ L	On breast 9 mos.
705	M	Screaming	16.8	26½	99	....	Bottle	E	Breast 3 weeks
23	F	Bronchitis	16.3	27½	97.8	76	.....	F	
688	?	Coryza	9.10	....	96.4	87	Bottle	F	Breast 1 month; age, 11½ months
454	M	Screaming	19.8	27½	96.2	79	.....	F	
758	M	Second degree of scalds of leg and thigh (left)		Well developed			Bottle	V ↑ L ↓ R	

Age, 1 Year; Normal Weight, 21 Pounds; Length, 27 Inches

199	F	Scabies	17.15	27	97	79	.....	E	
583	?	Wasting	19	28½	99.8	83	Bottle	V ↑ R ↓ L	Breast, 2 months
678	?	.....	15.13	....	98.4	80	Bottle	F	Breast fed 2 weeks (see also under 6 weeks F)
681	?	.....	17.15	....	98	83	Breast	V ↑↓ each	During sleep (see also under 6 wks. F)
765	M	External strabismus due to myopia		Normal development			Breast	F	

Age, 1 Year 1 Month; Normal Weight, 22 Pounds

13a	M	Wasting; phimosis	17	29½	98.4	74	.....	F	
16	F	Wasting	15.10	25	103.8	83	.....	F	
232	F	Wasting	14.4	29	98.4	80	.....	F	
368	M	Wasting	14.10	25½	98.8	82	.....	V ↑ R ↓ L	
434	F	Bronchitis	21.4	23¼	97.8	80	.....	F	
502	F	Screaming	?	?	?	?	.....	V ↑↓ each	Fortn'ght premature; mother suffered from albuminuria of pregnancy
621	F	Nil	19.12	31	97.2	91	Breast	V ↑ R ↓ L	
637	F	Screaming and wasting	10.13	23½	97.2	....	Bottle	E	Breast fed 5 weeks
646	F	Diarrhea and vomiting	18.6	29½	97.4	....	Breast	E	

PROTOCOLS OF CASES

Age, 1 Year 1 Month; Normal Weight, 22 Pounds—(Continued)

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
648	F	Diarrhea and vomiting	18.6	29½	97.4	....	.....	E	
652	F	Supernumerary thumb, right hand	12.2	22	98	....	.....	E	
700	F	Umbilical hernia	20.0	30½	99.2	....	Breast	E	During sleep

Age, 1 Year 2 Months; Normal Weight, 22½ Pounds

336	F	Umbilical hernia	21.8	29	98.4	80	.....	F	
354	M	Wasting	15.8	27	99.6	79	.....	E	
467	?	Rickets	16.9	29½	98.4	83	.....	F	
472	M	Bronchopneumonia	23.10	....	102.2	85	.....	F	
595	M	.....	16.3	26	99.4	70	.....	F	During sleep
714	F	Umbilical hernia; convulsions when 6 weeks old	20	29	98.6	87	Breast	V ↑ L ↓ R	During sleep; breast fed 10 months

Age, 1 Year 3 Months

137	F	Weakness of neck muscles; (?) slight imbecility	17.4	26	99	77	.....	F	
189	F	Loss of appetite	18.12	....	98.2	77	.....	F	
260	M	Intestinal toxemia	12.2	22½	98	82	.....	F	
504	M	Phimosis; right and left inguinal hernia	21.4	33½	97.6	80	.....	F	

Age, 1 Year 4 Months

396	M	Bronchitis and wasting	14.8	23½	98.6	80	.....	F	
489	M	(?) tuberculosis peritonitis	16.5	27½	98	87	.....	F	
736	F	Refusing food	20.11	30½	102	83	Bottle	F	Breast fed 6 weeks

Age, 1 Year 5 Months

381	F	Abscess of buttock	18.12	27	98.8	87	.....	V ↑ L ↓ R	8 months' fetus
587	M	Measles	17.10	23½	99	80	Breast	V ↑ L ↓ R	
589	F	Otorrhea	16.12	28½	99.2	83	Bottle	E	

Age, 1 Year 6 Months

17	M	Diarrhea and vomiting	14.8	29	100	83	.....	F	
18	M	Diarrhea; adenoids; perforation of right membrana tympani; anemia	18.7	31	98	80	Breast	F	
131	M	Eczema	19.10	31½	98	83	.....	F	
592	F	.....	20.15	29	98.4	77	Breast	E	
597	F	Malnutrition	16.15	27	98.2	70	.....	F	
671	M	Bronchopneumonia	18.5	31	99	87	.....	F	

Age, 1 Year 7 Months

76	M	Wasting; (?) tuberculosis	18.9	30¾	97	77	.....	F	Family history of tuberculosis
195	M	Wasting; (?) tuberculosis	....	32	98.6	84	.....	V ↑ R ↓ L	
435	M	Scabies	20.1	30	97.4	75	.....	F	Hydrocephalus

Age, 1 Year 8 Months

313	F	Pertussis	18.4	30	98.8	78	.....	F	
480	M	Pertussis	....	32	99.4	80	.....	F	
420	F	Bronchitis	24.2	28¾	97.8	78	.....	F	

Age, 1 Year 9 Months

94	F	Not taking well	22.10	32½	99	80	.....	F	
196	F	Well developed	.....	....	....	87	.....	F	
278	M	Slight bronchitis	20.4	31	98	84	.....	F	
625	M	.....	.....	....	....	....	.....	E	

PROTOCOLS OF CASES

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Feeding	Toe	Remarks
Age, 1 year 10 Months									
218	M	.....	.....	....	....	....	.....	F	
382	F	Congenital imbecility; no history of fits or imbecility in family	15.15	24½	99.2	79	.....	E	Fall on head at 10 months; family history of tuberculosis; breast fed 1 yr. 8 mo.
721	F	Bronchopneumonia; pertussis	Well developed		102.2	....	.....	E	
Age, 1 Year 11 Months									
87	F	Screaming	.....	35¼	98	84		F	
135	M	Wasting	13.4	26½	98.6	83		F	
492	M	Otorrhea	?	Well developed		88		V	Breast fed 9 months; pneumonia at 7 months
Age, 2 Years									
316	M	Malnutrition	18.12	....	98.4	87		F	
317	M	Debility and fretfulness	21.10	32½	97.8	78		F	8 months' fetus
346	M	Weakness of legs and ankles	.....	34¾	98.4	81		F	
369	F	.....	22.6	....	99.2	86		F	
546	?	Earache and occasional right sided convulsions	23.4	34½	98.4	84		F	
640b	F	Bronchitis	Normal development					F	
Age, 2 Years 2 Months									
443	M	.....	.....	31	98	81		F	Ringworm
569	M	.....	.....	30	98	77.7		F	
Age, 2 Years 6 Months									
302	M	.....	22.3	30	100	76		F	Pyrexia: child still unable to walk; no signs of nervous disease (e.g. paralysis, etc.)
402	M	.....	.....	37	97	75		F	
702	M	.....	.....	36	98.2	....		F	Breast baby
750	F	.....	.....	34½	98.2	....	V	↑ R ↓ L	Breast fed
Age, 2 Years 8 Months									
251	M	.....	.....	31¼	98.6	80	V	↑ R ↓ L	A case of petit mal: family history of fits; all reflexes normal, no Chvostek sign; intelligent infant; walked at 1 yr. 6 mo.; talked at 2 years
764	F	Scabies	.....	....	....	....		F	Breast 1 year
Age, 2 Years 9 Months									
285a	M	.....	23.1	30½	99.2	80		F	Left anterior poliomyelitis since 1 year old
640d	M	.....	.....	....	....	....		F	
Age, 2 Years 10 Months									
725	F	.....	.....	....	101	....		F	
Age, 2 Years 11 Months									
686	F	.....	23.1	34½	98.4	80	V	↑ R ↓ L	Pulmonary tuberculosis; breast fed
241	M	.....	97.6	....	....	77.7		F	Case of acute lymphatic leukemia*

* Differential blood count: polymorphonuclears, 9 per cent.; large lymphocytes, 71 per cent.; small lymphocytes, 7 per cent.; lymphoidocytes, 11 per cent.; myeloblasts, 2 per cent. Although from the point of view of the present investigation it has no special interest, it is a very instructive one from the general medical standpoint. The presence of lymphoidocytes in the blood is believed to point to an early fatal termination and, indeed, this child died suddenly within a couple of days after it was first seen by me.[52]

PROTOCOLS OF CASES

Age, 3 Years

Case	Sex	Complaint	Wt., Lbs.	Length, Inches	Temp.	C. I.	Toe	Remarks
466	?	.....	.....	34	100.2	80	F	
640c	M	.....	.....	....	....	....	F	Adenoids
722	F	.....	.....	....	98	....	F	

Age, 3 Years 4 Months

415	M	Injury left ankle	.....	....	....	....	V ↑ ↓ ↓ R (Left)	Synovitis, (?) tuberculosis

Age, 3 Years 5 Months

766	F	Urticaria	Normal development				E outer sole F inner sole	

Age, 3 Years 9 Months

337	F	General debility	.....	38¾	98	80	F	
759	F	Pain left knee	.....	....	....	....	E, F	(?) Rheumatic soft systolic bruit at apex

Age, 4 Years

618	F	Pertussis	.....	....	....	....	E	Convulsions at 11 a. m.; observation of reflex at 8 p. m.

Cases Over 4 Years

No.	Sex		Toe	Remarks
723	F		F	
724	F		F	Age 4½ years; myocarditis after diphtheria
770	M		F	Age 4 years 8 months
764a	F		F	Age 4 years 10 months
602a	M		F	Age 5 years
617	M	V	↑ R, ↓ L	Age 5 years 6 months
757a	M		F	Age 5 years 6 months
771	F		F	Age 5 years 6 months
772	M		F	Age 6 years
773	F		F	Age 6½ years
775	F		F	Age 6 years 9 months
774	M		F	Age 7 years 9 months
753	M 6 st. 4 ft. 6		E	Very typical Babinski phenomenon; complaint, walking on tip toes from infancy; age 9 years 4 months (see p. ....)
777	M ? st. 2-3 ft. 6	V	↑ R, ↓ L	Age 6 years (same case as 617)

CAN YEAST BE USED AS A SOURCE OF THE ANTINEURITIC VITAMIN IN INFANT FEEDING? *

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IOWA CITY

The reports of Eddy and Roper [1] and Daniels and Byfield [2] on the effect of adding substances containing the antineuritic vitamin [3] to the diets of artificially fed infants suggests that at least part of the difficulty in feeding these babies is due to a too low concentration of the antineuritic vitamin in many of the feeding mixtures. Cow's milk has been shown to contain less of this vitamin than many other natural foods,[4] the amount present being dependent on the vitamin content of the food of the cow, for it appears that "vitamins pass into the milk only as they are present in the diet of the mother." [5] When cow's milk is diluted, a necessary procedure in feeding young babies, unless some antineuritic vitamin is added from other sources, too little may be present in the feeding mixtures. The baby who is well and able to take his normal quota of food appears to get enough of this particular growth material to meet his needs. But the young baby or the baby with disturbed digestion who cannot take the usual amount of food may fail to gain because the antineuritic vitamin content of the food falls below his minimal requirement.

Eddy and Roper found that a specially prepared powder, made from the alcoholic extract of the pancreatic gland of lambs, when added to the diet of marasmic infants, stimulated growth. Previous experiments on rats had demonstrated that this powder contained the antineuritic vitamin. Daniels and Byfield, working with well babies, observed that many who were not gaining on food mixtures containing adequate amounts of the well known food constituents—protein, fat, carbohydrate and inorganic salts—increased in weight when the antineuritic vitamin (obtained from wheat embryo) was added to the feeding mixtures. In many instances these children were receiving food mixtures supplying from 115 to 130 calories on their theoretical weights—considerably more

* Received for publication, Aug. 18, 1921.
* This study was made possible through the courtesy of the Pediatric Department of the College of Medicine of the University of Iowa.

1. Eddy, W. H., and Roper, J. C.: Am. J. Dis. Child. **14**:189 (Aug.) 1916.
2. Daniels, A. L., and Byfield, A. H.: Am. J. Dis. Child. **18**:546 (Dec.) 1919.
3. The term antineuritic vitamin is used throughout the report to indicate the growth promoting vitamin designated as "Water Soluble B" by McCollum and his co-workers.
4. Osborne, T. B., and Mendel, L. B.: J. Biol. Chem. **34**:573, 1918.
5. McCollum, E. T.; Simmonds, N., and Pitz, W.: J. Biol. Chem. **27**:33, 1916.

than is usually considered necessary. Growth in a number of instances continued until somewhat less than 100 calories per kilogram was being given. These observations suggest a possible explanation for the well known fact that the baby fed artificially needs more food than the breast fed baby.

In view of these findings it seems desirable to obtain some inexpensive, readily available source of this antineuric vitamin which may be added to the food of the very young infant or the baby who for one reason or another is unable to take a sufficient amount of food to ensure an adequate ingestion of the vitamin. Neither the wheat embryo extract used by Daniels and Byfield, nor the pancreas preparation of

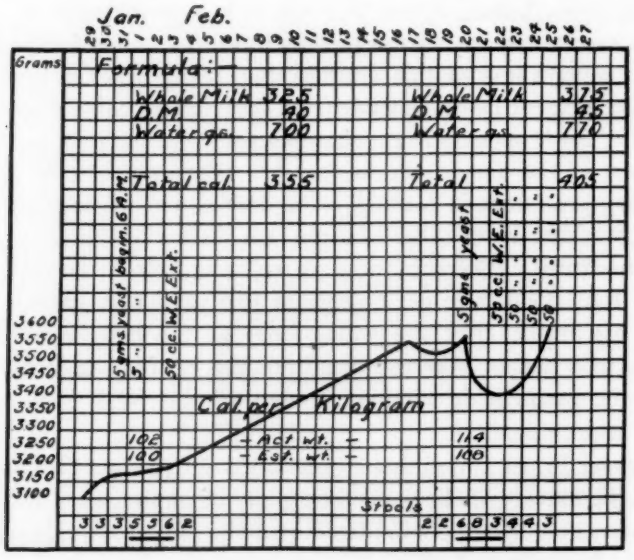

Fig. 1.—Sybil T.; born Dec. 21; birth weight, 2.858 gm.; aged, 1¼ months.

Eddy and Roper are easily available to the practitioner or the mother. The vegetable soup suggested [6] by the former investigators as a source of the antineuritic vitamin has been used with much success for well babies over 4 months of age. It is, however, not well tolerated by the very young baby or the baby suffering from digestive disturbances. Both orange [7] and tomato [8] juice can be used as sources of the antineuritic vitamin, but considerably more must be used than is necessary to protect against scurvy; and the young baby or ill baby frequently cannot take enough to furnish the required amount.

6. Daniels, A. L., and Byfield, A. H.: loc. cit.
7. Byfield, A. H., and Daniels, A. L.: Am. J. Dis. Child. **19**:349 (April) 1920. Osborne, T. B., and Mendel, L. B.: J. Biol. Chem. **42**:465, 1920.
8. Osborne, T. B., and Mendel, L. B.: J. Biol. Chem. **41**:451, 1920.

In animal experimentation yeast [9] has been used with much success as a means of furnishing this growth promoting vitamin, its potency in this respect being considerably greater than that of many foods tested,[10] especially milk.[11] Osborne and Mendel working with rats, observed that normal growth could be secured when 0.2 gm. per day was used as the only source of the vitamin, whereas 16 c.c. of milk was necessary to furnish an adequate amount. The possibility of using yeast as a means of increasing the antineuritic content of infants' food was suggested. The introduction of purin forming substances into the diets of infants did not commend itself, but since in all probability only a small amount of yeast would be needed to produce the desired results, it

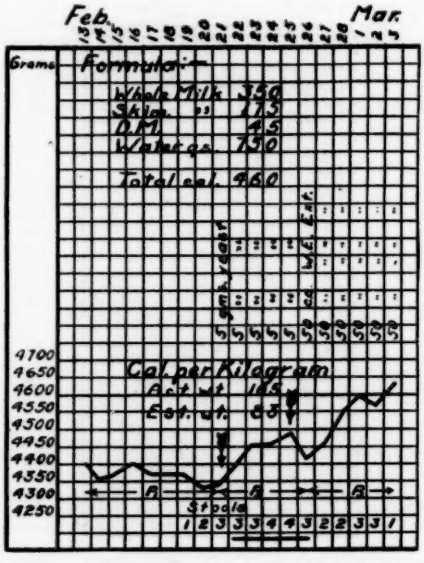

Fig. 2.—Clarice P.; born October 27; birth weight, 3,750 gm.; aged, 3½ months.

was reasoned that the good which might accrue would perhaps outweigh the theoretical objection. In the investigation from 2 to 10 gm. air dried pulverized yeast (Fleischmann's) were soaked in a small amount of water, subsequently boiled and added to the day's feeding. In certain instances, when no gain in weight resulted from the yeast additions, wheat embryo extract (50 c.c.) was substituted for the yeast, in order to make sure that the failure to gain was not due to a lack of the antineuritic vitamin. In some cases the wheat embryo extract was added for therapeutic purposes.

9. Osborne, T. B., and Mendel, L. B.: J. Biol. Chem. **31**:149, 1917.
10. Osborne, T. B., and Mendel, L. B.: J. Biol. Chem. **32**:309, 1917.
11. Osborne, T. B., and Mendel, L. B.: J. Biol. Chem. **34**:537, 1918.

In determining the influence on growth of a given vitamin addition to the diet of infants, several factors must be considered. The babies under observation should be as nearly normal as possible—that is, there should be no elevated temperatures, diarrhea, or other indications of indigestion or illness. The amount of food given must be sufficient to cover the physiologic requirements for age and weight. The feedings must be prepared with the greatest accuracy, as to amount of materials used and method of preparation. Care must be taken to see that all the food is taken and retained. It is most desirable that a stationary weight period of at least four days on a satisfactory food mixture should precede the period during which the added material is given. The

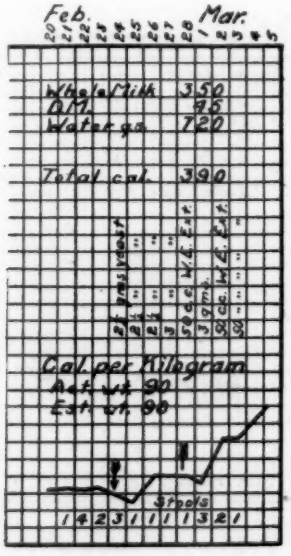

Fig. 3.—Keith M.; born December 27; birth weight, 3,595 gm.; age, 7 weeks.

character and number of the stools, both before and during the observation period, should be noted. If possible, the baby should be kept on the same formula, without the added vitamin, for four days following the period.

In testing the influence of yeast additions on growth, these methods, in so far as possible, were followed. The sixteen babies used for the observations, with the exception of two suffering from sudamina, were normal healthy babies from 5 weeks to 6 months of age. Details of the feeding mixtures, weights, etc., in a number of the more typical cases are given in the accompanying charts.

The most noticeable general effects of the yeast additions, especially with the younger babies, was the change in the number and character of the stools, a formed "safe" stool often becoming diarrheal. In many

instances not only was the character of the stool changed, but the number per day was greatly increased even when comparatively small amounts of yeast were used. These frequent diarrheal stools were in a number of cases followed by sudden losses in weight. The results were sometimes so disastrous that it was necessary to institute corrective measures at once. For example, the addition of 5 gm. dried yeast (equivalent to about two-thirds of a fresh yeast cake) to the feedings of Sybil T., a 6 weeks old baby, was followed by frequent watery stools. After three feedings (twelve hours) it was necessary to change to an "eiweiss" milk formula to check the diarrhea. Three weeks later the yeast additions were again tested with results that were even more

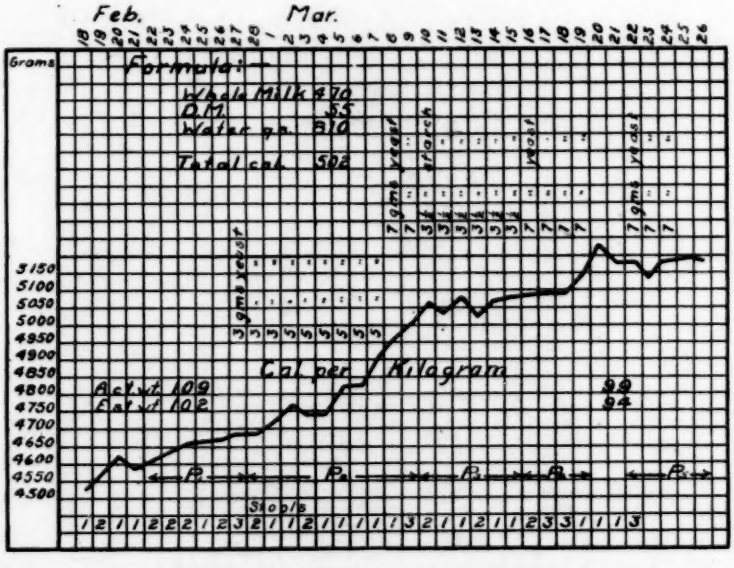

Fig. 4.—Gretchen S.; born Nov. 30, 1920; birth weight, 3,396 gm.; age, 2½ months.

marked than during the first yeast period. At this time the feedings were not well retained, and the stools increased from two per day to six and eight, respectively. There was a sudden loss in weight, and after a twenty-four hour trial the baby was returned to its previous formula to which were added 50 c.c. of our wheat embryo extract.

The addition of 5, 7 and 2½ gm. yeast to Robert L.'s feedings produced somewhat similar results—marked diarrhea and loss in weight when the larger amounts of yeast were used. With the smaller amount of yeast—2½ gm.—the stools were less frequent and formed, but apparently not enough of the antineuritic vitamin had been added with this small amount of yeast to produce any apparent physiologic effect; the subsequent addition of 50 c.c. of wheat embryo extract resulted in

an increase in weight. It would seem that in this case any good effects that might have accrued from the antineuritic content of the larger amounts of yeast were counteracted by the purgation produced.

The amount of the antineuritic vitamin necessary to stimulate growth in any given case depends obviously on the amount contained in the feeding mixture. In certain instances, it may be possible to use yeast with no untoward results, provided the antineuritic vitamin content of the food is only slightly below the minimum requirement, and, therefore, only a small additional amount is necessary. In the case of Robert L. and Keith M., 2½ gm. was not sufficient to produce any noticeable effects on growth, whereas the addition of 2½ gm. to the

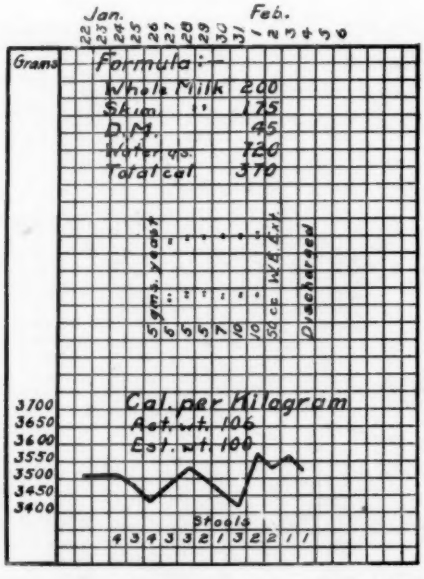

Fig. 5.—Walter A.; born Dec. 12, 1920; birth weight, 3,262 gm.; age, 6 weeks.

diet of Leona L. caused a slight gain in weight—60 gm. in five days—in spite of frequent stools. The substitution of 50 c.c. of wheat embryo extract, however, resulted in an increase of 190 gm. in five days. Similar increases were produced by the addition of wheat embryo extract in the other cases where yeast had been tested. In view of the marked effect of the yeast on peristalsis, in none of these children could larger amounts of yeast have been used.

With babies suffering from constipation, yeast, possibly, may be used with some degree of success, although we believe that there are better methods of meeting this problem. In four of our cases constipated stools became normal in consistency during the yeast additions. In one case (Gretchen S.) not only was the constipation relieved but

there was a considerable increase in weight. The addition of 3.5 and 7 gm. yeast, respectively, was followed by a weight increase of 370 gm. in eleven days. When yeast was omitted and starch (3 gm.) equivalent to the amount carried in the 7 gm. yeast was substituted, the weight became stationary, indicating that the gain was due to the antineuritic vitamin of the yeast rather than to the starch contained therein. A subsequent addition of yeast produced a slight gain in weight—50 grams in 5 days. At this time the low caloric value of the food—94 grams per kilogram, in all probability accounts for the small gain during the second yeast period.

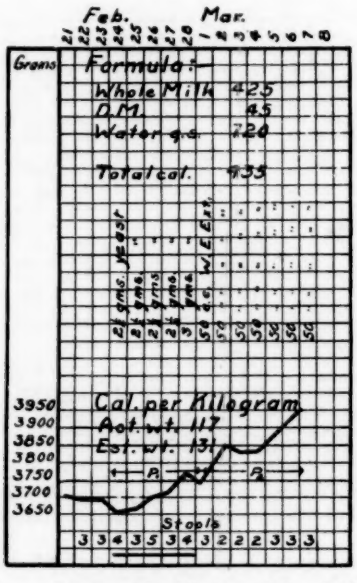

Fig. 6.—Leona L.; born January 8; birth weight, 3,040 gm.; age, 6 weeks.

At the time our other studies were being made there were in the hospital two rather severe cases of sudamina. Since Hawk [12] and his co-workers had obtained such good results with Baker's yeast in furunculosis, acne and other skin infections, it seemed desirable to test the effects of yeast on these two babies. In one case the infection had been running for about ten days, in the other case a somewhat shorter time. With both babies fairly large amounts of yeast—from 5 to 10 gm.—were used. During the period our staff were questioned each morning with regard to the condition of the babies. For a time several of them did not know that any treatment was being tried, so that their opinion

12. Hawk, P. B.; Knowles, F. C.; Rehfus, M. E., and Clarke, J. A.: J. A. M. A. **69**:1243, 1917.

was quite unbiased. There appeared to be no untoward results during the yeast additions, nor was there any apparent beneficial effects on the course of the infection. After days of yeast therapy the boils persisted. Indeed, in one other case (R.L.) boils developed during yeast feeding. The boils finally yielded in three days to the application of a dilute solution of iodin and benzol. In the cases noted there was no increase in weight, although fairly large amounts of yeast were used; nor was any diarrhea produced. The subsequent addition of 50 c.c. wheat embryo extract to the feedings was also without effect, indicating that stationary

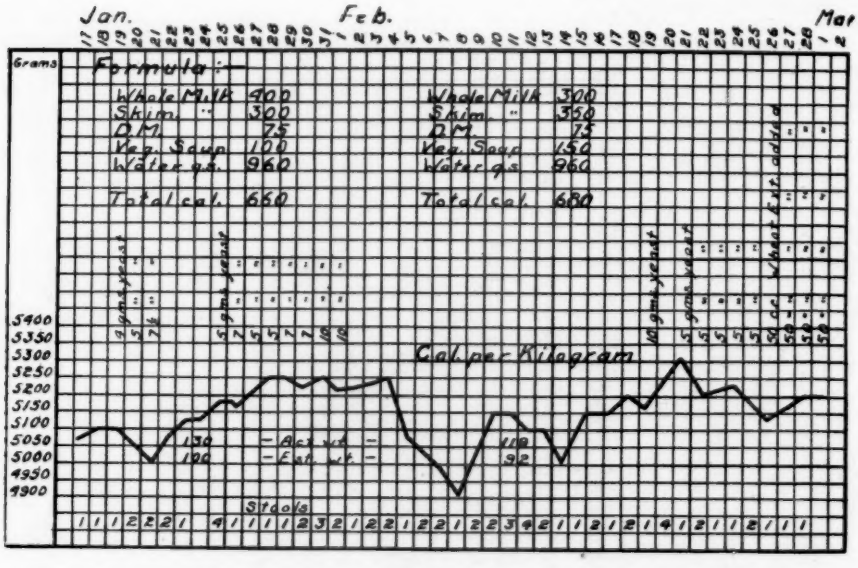

Fig. 7.—Robert G.; born July 10, 1920; birth weight, 3,247 gm.; age, 6 months.

weight in these cases was not due to a lack of the antineuritic vitamin. A failure to gain during an infection of this kind has been observed frequently.

Altogether, our experience with yeast points to the conclusion that it should not be used as a means of increasing the antineuritic content of infants' foods. All the babies under observation were well babies—that is, well from the standpoint of digestion. The addition of yeast, even in small amounts, resulted in diarrhea in a large number of cases, the untoward effects with the younger babies being more marked than with the older ones. Since in all probability the artificially fed baby most in need of some additional antineuritic vitamin will be the young baby, or the baby suffering from disturbed digestion, it is obvious that some substance other than yeast must be found as a means of supplying this.

A recent paper by Ladd [13] on the effects of yeast in infant feeding, published since the above report was written, corroborates our findings in certain respects. The details of the investigation are not given, therefore, critical comparisons cannot be made. Ladd, however, was unable to obtain evidence that yeast had any therapeutic effect on the course of furunculosis in infants; nor was there any evidence in his studies that yeast in the amounts given influenced the rate of growth. In one case fermentative diarrhea developed soon after the yeast was begun.

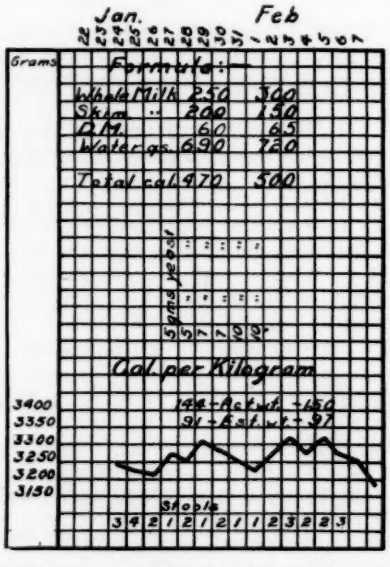

Fig. 8.—Wilbur Van H.; born Oct. 5, 1920; birth weight, 3,170 gm.; age, 3½ months.

REPORT OF CASES

Case 1.—*Clarice P.* The stimulation of growth during Period 2 was apparently due to the yeast additions, since it followed a period of stationary weight. During the yeast period the stools became watery and more frequent. The substitution of 50 c.c. of wheat embryo extract (Period 3) resulted in a gain of 230 gm. in five days, against an increase of 150 gm. in the five day yeast period.

Case 2.—*Robert L.* The addition of 5 and 7 gm. yeast, respectively, to the diet of Robert L., a baby who had always been a difficult feeding case, caused an increase in the number of stools and a subsequent loss in weight. Two and one-half grams were without effect on growth. The addition of 50 c.c. of wheat embryo extract resulted in an increase in weight.

Case 3.—*Walter A.* Yeast produced no untoward results nor did it stimulate growth. Apparently, his failure to gain was due to some cause other than a lack of the antineuritic vitamin.

Case 4.—*Julia McC.* The addition of 3 gm. yeast resulted in a temporary gain in weight. The increase in the number of diarrheal stools made it necessary to discontinue the yeast.

13. Ladd, M.: Arch. Pediat. **38**:423 (July) 1921.

CASE 5.—*Leona L.* The addition of 2.5 gm. yeast produced a slight gain in weight (Period 1). This, however, was accompanied by an increase in the number of stools. The substitution of 50 c.c. wheat embryo extract (Period 2) was more effective than the yeast. During this period the stools became normal.

CASE 6.—*Walter Van H.* was suffering from a severe case of sudamina. The addition of yeast in fairly large amounts—5, 7 and 10 gm., respectively—was without effect on growth; nor did the yeast appear to affect the course of the infection, which was subsequently cleared up after three days' treatment with a dilute solution of iodin and benzol.

CASE 7.—*Mary B.* The addition of 5 gm. yeast (dry weight) to the diet of this 7 weeks old baby produced diarrhea, manifested by many loose, watery stools.

CASE 8.—*Sybil T.* The addition of 5 gm. yeast produced severe diarrhea in a baby, 6 weeks old. Similar results were obtained three weeks later. At both times the effects were so marked that it was necessary to discontinue the yeast feedings.

CASE 9.—*Gretchen S.* The addition of yeast to the diet apparently stimulated growth without producing the untoward results observed in many of our other babies. This baby, however, was always quite constipated. The yeast additions were followed by soft yellow stools. During Period 3 the effect of 3.5 gm. of arrow starch (the amount contained in the yeast used) was tested. The weight remained stationary, indicating that the gain in Periods 2 and 4 were the results of the yeast additions. During Period 5 the caloric value of the food (94 calories per kilo on her theoretical weight) was too low to admit of gain in weight.

CASE 10.—*Keith M.* The addition of 2.5 and 3 gm., respectively, of dry yeast to the milk feedings produced no gain in weight. The effect on the stool was not marked. The substitution of 50 c.c. wheat embryo extract for the yeast resulted in a subsequent gain in weight.

CASE 11.—*Robert G.* The addition of yeast to the diet was without influence on growth. It also appeared to have no effect on a marked condition of sudamina. This baby apparently was not "well born" and had been a difficult feeding case from the first. The failure to grow was not caused by a lack of the antineuritic vitamin, as shown by the fact that the addition of 50 c.c. of our wheat embryo extract was also without effect.

THE INCIDENCE OF PROTEIN SENSITIZATION IN THE NORMAL CHILD*

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Although an extensive literature has accumulated on the diagnostic significance of protein sensitization tests in certain disease conditions, no attempt has hitherto been made to examine a large number of normal children for possible potential anaphylactic cases; nor have we been able to find any comparison between the incidence of protein sensitization in a large number of normal and abnormal subjects. The present widespread general interest in protein sensitization, first clinically and conclusively demonstrated by Schloss[1] and later by the voluminous and epochal researches of Walker,[2] suggested the necessity of this work.

It is the aim of this paper to show:

1. The incidence of protein sensitization in the normal child.
2. The relative frequency of sensitization to the specific protein in relationship to age and diet.
3. The relative frequency of sensitization to horse serum in those who have and those who have not been injected with prophylactic sera of any kind.

The children of this series were mostly Jewish, living on the East Side of Harlem and ranging in age from 2 to 15 years. The sexes were about evenly divided. A consideration of the diet of these children is necessary for a correct interpretation of the various protein reactions recorded. Of the cereal grains, wheat, corn, rice, rye, oats and barley are most common; it is interesting to note that the bread is practically without exception mostly rye. Eggs, milk and cheese are used freely. The meats most frequently eaten are beef and chicken, occasionally lamb and veal and not infrequently goose. Herring, smoked salmon, pike, smelts, and white fish are consumed often and in considerable quantities. Nearly all the different vegetables are used, especially potatoes and peas. As a rule, this class of children does not regularly indulge in vegetables and they are not encouraged to eat them. Fruits are eaten in season. Nuts are eaten occasionally. Milk, coffee and tea are indulged in freely.

Great care was exercised in the selection of the children to be tested. Each child's history was inquired into carefully and a thorough

* Received for publication, Sept. 15, 1921.
* From the Children's Clinic, Mount Sinai Hospital, New York.
1. Schloss, O. M.: Am. J. Dis. Child. **3**:341 (April) 1912.
2. Walker, I. C., and Adkinson, J.: J. M. Research **32**:287, 1917.

physical examination made. Only those presenting negative histories and physical evidence of a possible or probable anaphylaxis were tested. The children considered anaphylactic in origin were as follows: Bronchial asthma, recurrent bronchitis, seasonal and perennial hay fever, periodic vomiting, gastro-enteritis, acute and chronic eczema, urticaria, dermatoses, erythema multiforme, angioneurotic edema, iritis and conjunctivitis.

Selection of Protein.—It is extremely important to select the proper material, especially in a series of this character. The protein must be readily soluble and contain all the proteins composing the food.[3] The activity of most of the proteins was assured by definitely positive reactions in known anaphylactic cases. The protein dilutions were prepared fresh every five days and kept in well corked vials in the icebox. For use the protein was dissolved in tenth normal sodium hydroxid in a strength of 1 : 50.

Technic of the Tests.—The cutaneous scratch test is the one solely employed. After the flexor surface of the forearm has been cleaned with alcohol, linear cuts about ⅛ inch long and 1½ inches apart are made with a sharp scalpel. The cuts should be deep enough to penetrate the outer layers, without drawing blood. While blood does not interfere with the test, we consider it poor technic and unnecessary. Enough cuts are made at one time for all the proteins to be tested, allowing two for controls. The location of these controls is important; one is near the bend of the elbow where the skin is more sensitive, the second control is placed near the wrist, the tests are made between these two points. On each cut is placed a drop of the test solution. One drop of the tenth normal sodium hydroxid is placed on the controls. After the dissolved protein has remained in contact with the cuts for one half hour, it is wiped away and the reading made. The reactions are always to be compared with the controls. During the test, the proteins must be kept in solution and drying prevented by the addition of a drop tenth normal sodium hydroxid.

If a reaction appears within a few minutes and rapidly becomes larger accompanied by an intense erythema and itching, it is well to wipe away the protein and thus prevent further absorbtion and the disagreeable symptoms of a possible acute anaphylaxis. It must be remembered that even cutaneous tests are not without danger of inducing anaphylactic shock.

Skin Irritability.—If the skin of a patient shows evidence of irritability, this fact should be noted for the proper final interpretation. The less the skin is traumatized, the less the danger of irritation. The

3. All proteins we used were prepared by the methods and under the supervision of R. P. Wodehouse.

manifestations may be in the form of wheals of different sizes, varying degrees of erythema, or both. These pseudoreactions may appear almost immediately after the cuts are made and be present at the time the true reactions are read. At times cutaneous irritability disappears within from five to ten minutes after the cuts are made, thus not interfering with a proper final reading. At other times it does not begin to appear until from fifteen to twenty minutes after the tests are made. The controls in such cases are very important guides. It is best to make a single row of tests down the forearm about two inches apart and if the tests are repeated, little or no irritability is encountered, enabling us to interpret the end-results more easily and with greater certainty.

Types of Reaction.—Due to a lack of standardization of technic and classification of the reactions, much of the statistical data presented in the literature is more or less confusing, which tends to distract from its value. The technic in our series was carried out by one of us (M. M. P.) thus assuring uniformity of procedure.

The classification we adopted is essentially the one employed by Walker [2] and is briefly as follows:

1. Positive reaction: (a) Distinct urticarial wheal, 0.5 cm. or more in diameter, surrounded by a zone of erythema in the presence of a negative control. The wheal is always irregular in outline, caused by edema spreading along the lymph spaces.

(b) Distinct urticarial wheal, less than 0.5 cm. in diameter, surrounded by a zone of erythema of two or more cm.

(c) Only a zone of erythema of two or more cm., with little or no elevation about the cut.

2. Doubtful reaction: All reactions measuring less than the above and more than the control.

3. Delayed reaction: This reaction is described by Walker as negative at the end of one half hour, but on the following day the cut appears like a slight infection; the surrounding skin is hot, very red and slightly elevated. Frequently the cut contains pus which is always sterile. This reaction was not noted in our series as the patients were not observed for the twenty-four hours following the tests.

4. Negative reaction: Those not differing in any way from the control.

The Cutaneous Versus the Intracutaneous Reaction.—We have adopted the cutaneous in preference to the intracutaneous test only after very careful and diligent comparisons. The same conclusion was reached by Schloss [4] who said:

4. Schloss, O. M.: Am. J. Dis. Child. **19**:433 (June) 1920.

During the past few years I have used the cutaneous test entirely as opposed to the intracutaneous test. This was done only after having made a series of tests by both methods on normal patients and on patients suffering from definite food idiosyncrasy. Such tests were made on 100 infants and children not apparently suffering from disturbances due to food, and in thirty who suffered from definite symptoms due to the ingestion of some variety of food. . . . The intracutaneous test is more sensitive. In three patients later proved to have mild idiosyncrasy to milk, intracutaneous tests proved positive while cutaneous tests were negative. This seems to be the only point in favor of the intracutaneous test.

Against it is the fact that it is apt to be misleading. Pseudoreactions occur which are difficult to interpret and in some instances patients give reactions which seem positive despite the lack of clinical evidence that the substance tested causes any symptoms. Many vegetable proteins are difficult to obtain in a form soluble in physiologic sodium chlorid solution, but are soluble only in alkaline solvents. Such solutions cannot be used for intradermal tests. To insure sterility, the proteins must be prepared carefully, which adds considerable technical difficulty. On two occasions I have seen severe infections due to such tests. Another objection is that a very sensitive patient may be made seriously ill by the injection of even a minute amount of the protein to which he reacts. In consideration of these objections it seems that, for general use the cutaneous test is the one to be chosen.

Walker [2] also concludes that:

The skin test is specific; it separates closely related proteins; it is sufficiently sensitive and yet not too sensitive; . . . it is easy to do, and it does not inconvenience the patient. The intradermal test is much less specific; it does not always separate closely related proteins, therefore it may be nonspecific; it is much too sensitive, therefore it is . . . very erratic; it is also more difficult to do, and it causes the patient considerable annoyance and discomfort. Therefore, it is not as practical when many proteins must be tested.

The Cutaneous Reaction in the Normal Child.—Formerly, when a patient with a positive or doubtful reaction developed symptoms resembling any of the now familiar anaphylactic states and the reaction was proved not to be the etiologic factor in the production of the symptoms, the phenomenon was considered an accidental protein sensitization of no significance. It is our belief that, when such reactions occur, these patients are fertile soil and always in danger of developing an anaphylactic condition. We maintain that a normal child exhibiting a skin reaction above the control is a potential anaphylactic case and should be regarded as such.

It must be borne in mind that these children are free from all anaphylactic symptoms because they have acquired a certain amount of individual tolerance against the specific protein causing sensitization. Symptoms develop only when the intake of that protein surpasses the threshold of tolerance. The limit of tolerance varies in different individuals as well as in the same individual at different times. Consequently, it is impossible to predetermine one's tolerance to proteins, or foretell the onset of anaphylactic symptoms by the degree of the skin reaction. A positive reaction does not necessarily predicate the onset of symp-

toms in one case while a doubtful reaction may augur a very violent attack. Two cases in point are the following:

History.—C. H., male, 5½ years of age, white, one of a family of eight, one of our series, was last tested in May, 1921. Fifty-two proteins were tested, of which the following showed sensitization: Horse dander, dog hair, cat hair and fig gave positive reactions; egg whole, egg white, cucumber, asparagus, sweet potato, buckwheat, chestnut, chicken feathers and tea gave plus minus reactions. Aug. 16, 1921, he returned to the clinic suffering from a typical attack of bronchial asthma; symptoms much worse at night.

Treatment.—The treatment instituted was a corrective diet, and when he returned two weeks later, he was free from his asthma and as well as ever.

History.—J. T., male, aged 14, white, not in our normal series, has been having attacks of bronchial asthma for the past four years. He was tested against seventy-three proteins consisting of food, serum, epidermal and bacterial. He gave a positive reaction to rabbit hair and a plus minus reaction to horse serum and lamb. These tests were repeated a number of times.

Sept. 16, 1921, he was given a prophylactic dose of 1 c.c. (1,000 units) diphtheria antitoxin [5] and within fifteen minutes he was seized with a severe attack of dyspnea lasting an hour, marked respiratory oppression and profuse expectoration; one-half hour later there developed a generalized erythema and giant urticaria with severe itching, edema of the hands, lips, cheeks and eyelids, marked vertigo, vomiting and cramps. September 17 he was entirely free from asthma, the rash had disappeared, but swellings still were present. He was tested with horse dander and horse serum which gave very distinct reactions. The reaction to rabbit hair was more intense than ever before. September 19 rabbit hair only gave a positive reaction; horse dander and serum, horse dander alkali metaprotein and horse dander peptone gave negative reactions.

Much of the sensitization occurring in children takes place through the intestinal tract. Talbot [6] found that "during infancy and childhood practically all cases of sensitization are due to foods." Because of adaptation or desensitization, allergy to food becomes less common as the age of puberty is reached. The author cites literature to show that the gastro-intestinal tract of the newly born infant is permeable to undigested foreign proteins, hence the infant develops the power of destroying such protein up to its limit of tolerance.

In 1913, Richet [7] stated that "experimental alimentary anaphylaxis is difficult to bring about under conditions of healthy digestion . . . because the individual passes a minimal quantity of unchanged albumin."

Vaughn [8] asserts that the sensitized group resulting from protein digestion is destroyed in normal digestion; that it is only under abnormal conditions that protein sensitization results through the alimentary tract.

Schloss and Worthen [9] found that the intestinal tract of normal infants is usually impermeable to undigested foreign protein. In

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5. Health Department, New York City.
6. Talbot, F. B.: Boston M. & S. J. **175**:409, 1916; **179**:385, 1918.
7. Richet, C.: Seventeenth Internat. Cong. Med., Lond., 1913, Sect. 4, Pt. 1, p. 13.
8. Vaughn: Protein Split Products, 1913.
9. Schloss, O. M., and Worthen, T. W.: Am. J. Dis. Child. **11**:342 (April) 1916.

agreement with other observers quoted by them, they also found that in nutritional or gastro-enteric disorders, foreign protein may be absorbed in an undigested or partially digested state and appear in the urine.

Thus comparing these findings with the evidence presented in Tables 1 to 15, one can understand how sensitization can readily occur in normal children who occasionally have some gastro-enteric disorder, and who acquire sufficient tolerance to their particular dietary to ward off symptoms of the anaphylactic state.

When that tolerance is exceeded, anaphylactic symptoms become evident. A positive reaction in the normal child may not have the same significance as a doubtful reaction in the abnormal child even though he shows multiple positive reactions.

C. T., a white male child, aged 10, suffering from repeated attacks of bronchial asthma for the past eight years (referred by Dr. Mary Rose) gave positive reactions to buckwheat, wheat glutenin, and globulin, potato, cattle hair and horse serum and doubtful reactions to oats, corn, rice, rye, lima beans, beans and horse dander. After careful study he was permitted to have all food except oatmeal, improvement followed immediately and was very evident two days after change in diet. This case distinctly shows tolerance for the positive-reacting proteins and not the doubtful reacting proteins.

Caulfield [10] has presented cases which show that it is possible for one to escape the clinical manifestations of anaphylaxis even though sensitized to proteins to which exposure is inevitable.

TABLE 1.—CASES TESTED FOR SENSITIZATION TO CEREAL GRAINS

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Wheat	24	100	76	200	0	2	98
Corn	24	100	76	200	0	0.5	99.5
Rice	24	100	76	200	0	0.5	99.5
Rye	24	100	76	200	0.5	8.5	91
Oats	24	100	76	200	0	1	99
Barley	24	100	76	200	0	1.5	98.5
Buckwheat	11	63	30	104	0	1.92	98.08

Considering the data presented in the present series, Tables 1 to 8 show the results of the various food proteins tested. In Table 1 the total number of doubtful reactions was thirty among twenty-six children, equally divided as to sex. The one positive reaction to rye occurred in a boy aged 12 years. It is interesting to note seventeen doubtful reactions to rye.

10. Caulfield, A. W.: J. A. M. A. **76**:1071 (April 16) 1921.

The total number of doubtful reactions to egg and milk (Table 2) were eleven occurring in six cases. There were no positive reactions.

TABLE 2.—Cases Tested for Sensitization to Eggs and Milk

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Egg, whole	19	111	74	204	0	1.96	98.04
Egg white	19	111	74	204	0	1.96	98.04
Egg yolk	19	111	74	204	0	0.98	99.02
Milk, cow's	19	111	74	204	0	0	100
Casein	19	111	74	204	0	0	100
Lactalbumin	19	111	74	204	0	0	100
Cheese	12	55	38	105	0	0.94	99.16

When the meat tests were conducted turkey and pork were used as controls, since they are rarely ingested by these children. Four cases showed doubtful reactions, equally divided between beef and veal (Table 3).

TABLE 3.—Cases Tested for Sensitization to Meats

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Beef	13	61	30	104	0	1.94	98.06
Chicken	13	61	30	104	0	0	100
Lamb	13	61	30	104	0	0	100
Veal	13	61	30	104	0	1.94	98.06
Goose	13	61	30	104	0	0	100
Turkey	13	61	30	104	0	0	100
Pork	13	61	30	104	0	0	100

Only one child shows a doubtful reaction to fish (Table 4); the control protein in this series was taken from the oyster.

TABLE 4.—Cases Tested for Sensitization to Fish

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Herring	12	55	38	105	0	0	100
Salmon	12	55	38	105	0	0	100
Pike	12	55	38	105	0	0	100
Smelts	12	55	38	105	0	0.94	99.06
Mackerel	12	55	38	105	0	0	100
Bluefish	12	55	38	105	0	0	100
Haddock	12	55	38	105	0	0	100
Oyster	12	55	38	105	0	0	100

Of nineteen vegetables tested only six gave a doubtful reaction (Table 5). These were asparagus, cucumber, lettuce, peas, white potato and sweet potato; four children showed the reaction.

TABLE 5.—CASES TESTED FOR SENSITIZATION TO VEGETABLES

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Beans	11	52	41	104	0	0	100
Asparagus	11	63	30	104	0	0.96	99.04
Beet	10	56	40	106	0	0	100
Cabbage	11	52	41	104	0	0	100
Carrot	8	60	37	105	0	0	100
Cauliflower	14	61	32	107	0	0	100
Cucumber	14	61	32	107	0	0.93	99.07
Celery	10	56	40	106	0	0	100
Lentil	10	56	40	106	0	0	100
Lettuce	10	56	40	106	0	0.94	99.06
Lima bean	14	61	32	107	0	0	100
Onion	10	56	40	106	0	0	100
Peas	11	52	41	104	0	0.96	99.04
Potato	11	52	41	104	0	0.96	99.04
Radish	14	61	32	107	0	0	100
Rhubarb	10	56	40	106	0	0	100
Spinach	10	56	40	106	0	0	100
Sweet potato	14	61	32	107	0	0.93	99.07
Tomato	10	56	40	106	0	0	100

When fruits were tested, one boy gave a positive reaction to plum, four others gave a doubtful reaction to fig (Table 6).

TABLE 6.—CASES TESTED FOR SENSITIZATION TO FRUITS

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Orange	8	60	37	105	0	0	100
Banana	8	60	37	105	0	0	100
Grape fruit	8	60	37	105	0	0	100
Strawberry	8	60	37	105	0	0	100
Peach	8	60	37	105	0	0	100
Pear	8	60	37	105	0	0	100
Pineapple	8	60	37	105	0	0	100
Cantaloupe	8	60	37	105	0	0	100
Fig	14	61	32	107	0	3.73	96.27
Plum	14	61	32	107	0.93	0	99.07
Raspberry	14	61	32	107	0	0	100
Blackberry	14	61	32	107	0	0	100

Only one child gave a doubtful reaction to nuts (Table 7). This sensitiveness was shown to chestnut.

TABLE 7.—CASES TESTED FOR SENSITIZATION FOR NUTS

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Almonds	11	52	41	104	0	0	100
English walnut	11	52	41	104	0	0	100
Peanut	11	52	41	104	0	0	100
Brazil nut	11	52	41	104	0	0	100
Chestnut	11	63	30	104	0	0.96	99.04

The common beverages, coffee, tea and cocoa, gave three doubtful reactions; two for coffee and one for tea; one child was sensitive to both (Table 8).

TABLE 8.—Cases Tested for Sensitization to Beverages

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Coffee	11	63	30	104	0	1.92	98.08
Tea	11	63	30	104	0	0.96	99.04
Cocoa	11	63	30	104	0	0	100

In Table 9 are presented the results of tests with the proteins from the epidermis of the horse, dog, cat, chicken and goose. The three positive reactions occurred in a boy aged 5½ years. Six doubtful reactions were observed in six children. The children who showed doubtful reactions to horse dander gave negative reactions to horse serum and had never received injections of any kind of serum.

TABLE 9.—Cases Tested for Sensitization to Epidermal Proteins

Protein	Number Tested				Percentage		
	2-5 Years	5-10 Years	10-15 Years	Total	Positive	Doubtful	Negative
Horse dander	19	111	74	204	0.49	1.96	97.55
Dog hair	11	63	30	104	0.96	0.96	98.08
Cat hair	11	63	30	104	0.96	0	99.04
Chicken feathers	11	63	30	104	0	0.96	99.04
Goose feathers	11	63	30	104	0	0	100

The incidence of sensitization to horse serum was determined by a more extensive series of tests to determine the relative safety of administering diphtheria antitoxin. The only child who gave a doubtful reaction to horse serum (Table 10) was a boy, aged 3½ years, whose history as to diphtheria was negative and who had never received an injection of any kind of serum.

TABLE 10.—Cases Tested for Sensitization for Horse Serum

Age	Number Cases	Number Positive	Number Doubtful	Number Negative
2	3	0	0	3
3	27	0	1	26
4	26	0	0	26
5	50	0	0	50
6	50	0	0	50
7	54	0	0	54
8	56	0	0	56
9	44	0	0	44
10	51	0	0	51
11	50	0	0	50
12	30	0	0	30
13	27	0	0	27
14	9	0	0	9
Total	477	0	1	476

Table 11 shows the interval between the injection of diphtheria antitoxin and the skin test for horse serum. Eighty children were tested and all reacted negatively. Four were injected twice as they had the disease twice.

TABLE 11.—Interval Between Horse Serum (Diphtheria Antitoxin) Injection and Date When Horse Serum Tests Were Made

Interval	No. Cases	Interval	No. Cases
1-6 months	2	6½ years	2
6 months	3	7 years	3
1 year	9	7½ years	1
1½ years	4	8 years	5
2 years	5	8½ years	3
2½ years	6	9 years	2
3 years	10	9½ years	1
3½ years	1	10 years	1
4 years	3	10½ years	1
4½ years	4	11 years	2
5 years	8	11½ years	2
5½ years	5	12 years	1
6 years	6		
Total			84

Table 12 gives the ages of the patients at the time of testing.

TABLE 12.—Age of Patients at Time of Testing for Horse Serum and Having Had Injections of Diphtheria Antitoxin

Age, Years	No. Cases	Age, Years	No. Cases
2	0	9	10
3	0	10	8
4	4	11	6
5	3	12	8
6	7	13	4
7	12	14	5
8	13		
Total			80

All positive reactions of the entire series are summarized in Table 13. There was none among the children aged from 2 to 5 years. Two boys in the five to ten year period gave four positive reactions; one boy in the from ten to fifteen year period was sensitive to rye. Thus in a series of 719 tests, there were five children who gave positive reactions. These occurred in three boys. The boy who was sensitive to horse dander, dog hair and cat hair gave a negative history and reaction to horse serum injection.

TABLE 13.—Summary of the Positive Reactions

Protein	2-5 Years		5-10 Years		10-15 Years		Total	
	Number Tested	Number Positive	Number Tested	Number Positive	Number Tested	Number Positive	Number Tested	Number Positive
Rye	24	0	100	0	76	1	200	1
Plum	14	0	61	1	32	0	107	1
Horse dander	19	0	111	1	74	0	204	1
Dog hair	11	0	63	1	30	0	104	1
Cat hair	11	0	63	1	30	0	104	1
Total	79	0	398	4	242	1	719	5

In Table 14 we have a summary of the total number of doubtful reactions, sixty-seven in a series of 4,274 tests.

TABLE 14.—Summary of the Doubtful Reactions

Protein	2-5 Years		5-10 Years		10-15 Years		Total	
	Number Tested	Number Doubtful	Number Tested	Number Doubtful	Number Tested	Number Doubtful	Number Tested	Number Doubtful
Wheat	24	1	100	2	76	1	200	4
Corn	24	1	100	0	76	0	200	1
Rice	24	0	100	0	76	1	200	1
Rye	24	6	100	10	76	1	200	17
Oat	24	0	100	2	76	0	200	2
Barley	24	1	100	2	76	0	200	3
Buckwheat	11	0	63	2	30	0	104	2
Egg, whole	19	0	111	3	74	1	204	4
Egg white	19	0	111	3	74	1	204	4
Egg yolk	19	0	111	2	74	0	204	2
Cheese	12	0	55	0	38	1	105	1
Beef	13	0	61	2	30	0	104	2
Veal	13	1	61	1	30	0	104	2
Smelts	12	0	55	1	38	0	105	1
Asparagus	11	0	63	1	30	0	104	1
Cucumber	14	0	61	1	32	0	107	1
Lettuce	10	0	56	1	40	0	106	1
Peas	11	0	52	0	41	1	104	1
Potato	11	0	52	1	41	0	104	1
Sweet potato	14	0	61	1	32	0	107	1
Fig	14	0	61	3	32	1	107	4
Coffee	11	1	63	0	30	0	104	1
Tea	11	1	63	1	30	0	104	2
Chestnut	11	0	63	1	30	0	104	1
Horse dander	19	1	111	0	74	3	204	4
Horse serum	56	1	254	0	167	0	477	1
Dog hair	11	0	63	1	30	0	104	1
Chicken feather	11	0	63	1	30	0	104	1
Total	477	14	2,314	42	1,483	11	4,274	67
							M	F
(2- 5 years) Total number ± reactions were	14		distributed among	9	patients		6	3
(5-10 years) Total number ± reactions were	42		distributed among	30	patients		18	12
(10-15 years) Total number ± reactions were	11		distributed among	9	patients		5	4
(2-15 years) Total number ± reactions were	67		distributed among	48	patients		29	19

The total number of children tested was 502. The total number of tests performed was 9,406. Seventy-four different proteins were used in these tests, averaging 18.73 proteins per case. It is interesting to note that rye (bread commonly eaten by these children) gave 3.38 per cent. doubtful reactions of the total actual number of cases tested.

The summary of the entire series is presented in Table 15.

TABLE 15.—Summary of Reactions in Relation to Actual Number of Cases Tested and Tests Performed

Age, Years	Actual Number of Cases Tested	Number of Tests Performed	Number of Positive Reactions	Per Cent. Cases With Positive Reactions	Per Cent. Positive Reactions to No. of Tests Performed	Number of Doubtful Reactions	Per Cent. of Cases With Doubtful Reactions	Per Cent. Doubtful Reactions to No. of Tests Performed	Cases With Positive Reactions	Cases With Doubtful Reactions
2- 5	58	1,008	0	0	0	14	15.5	1.38	0	9
5-10	270	5,136	4	0.70	0.07	42	11.11	0.81	2	30
10-15	174	3,262	1	0.57	0.03	11	5.17	0.33	1	9
Total	502	9,406	5	0.53	0.05	67	9.56	0.71	3	48

CONCLUSIONS

1. Ten per cent. of the children show doubtful and positive reactions which decrease as their age increases, indicating progressive desensitization. This coincides with Talbot's observations.

2. The incidence of sensitization in normal as compared with abnormal children indicates that the ratios are correct. To compare our incidence of sensitization with a pathologic series, we have found the report of Walker [11] the most complete. The author tabulates 400 cases of bronchial asthma and makes the following statements; four-fifths of the patients whose asthma began during infancy were sensitive; two-thirds of those whose asthma began during childhood were sensitive; one-half of those whose asthma began during young adult life were sensitive; one-fourth whose asthma began during adult life were sensitive, none were sensitive who began after middle life.

3. The foods giving doubtful and positive reactions are those frequently indulged in and compose an essential part of the dietary of these children.

4. The foods giving the greatest number of reactions in these normal children correspond proportionately with those in the abnormal children.

5. The administration of diphtheria antitoxin incurs practically no risk of anaphylaxis.

6. All reactions above the control are true protein sensitizations and should be regarded as potential anaphylactic cases.

7. It is necessary to institute prophylactic measures in normal cases presenting sensitization without symptoms.

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11. Walker, I. C.: A Clinical Study of 400 Patients with Bronchial Asthma, Canad. M. Assn. J. **9**:97 (Feb.) 1919.

AN UNUSUAL EXANTHEM OCCURRING IN INFANTS *

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During the past year I have observed several cases of an unusual nature occurring in infants. These cases were characterized by an acute illness, with high fever, lasting from three to four days. The temperature then fell quickly to normal, and with the subsidence of the fever an eruption appeared lasting from one to three days. The patients were all infants whose ages ranged from 9 months to 2 years. Only one member of a family contracted the disease. The following protocol of the first case observed during March, 1921, will serve as an example of this condition.

REPORT OF CASE

History.—Baby H. T., aged 9 months, became suddenly ill March 10, 1921. He had been perfectly well the day before and had not been out of the house for at least two weeks prior to his illness.

Physical Examination.—Examination showed a well nourished and developed infant, acutely ill and extremely irritable. There was no toxemia. The head was normal in shape and size and the anterior fontanel was level. There was no discharge from the nose. The ear drums were normal. The throat was slightly congested. The tongue was normal. No Koplik's spots were seen on the mucous membranes. There was no adenitis. The examination of the heart and lungs was negative, likewise the examination of the abdomen. The reflexes were normal. No eruption was seen on the skin. The temperature was 104 F.; pulse, 160; respirations, 35.

Laboratory Examination.—Urine was negative for albumin, sugar, casts and leukocytes. The white blood count was 5,400, with 85 per cent. lymphocytes and 15 per cent. polymorphonuclears.

The temperature remained between 103 and 104 F. for three days. During this time the patient was seen daily and examined carefully. No cause for the temperature was found. The infant seemed quite ill, but not toxic. Most of his feedings were refused.

Exanthem.—March 13, the fourth day of the disease, a telephone message was received from the mother stating that the baby's fever was down and he was much better, but that he had an eruption which she thought resembled measles. The temperature was 98 F. The baby was much less irritable and was taking his feedings well. No Koplik's spots were observed. The skin was covered with a discrete maculopapular eruption most marked on the trunk. However, there were some lesions on the face and extremities. The lesions were rose red in color and varied in size from a pin head to a large pea. There was no pruritis. The mother noticed that the eruption had first appeared on the neck and had quickly spread downward. The following day the eruption began to fade. The baby's condition was excellent. Two days later the eruption had completely disappeared. There was a very faint desquamation. A brother, aged 4, who was directly exposed to the baby throughout the illness did not contract the disease.

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* From the Department of Pediatrics and Infectious Diseases, University of Michigan Medical School, Dr. D. M. Cowie, Director.

Seven other cases were observed during the next four months. They all followed the same course, a period of fever for three or four days with a sudden drop in temperature coincident with an eruption lasting from two to three days and then disappearing. The first case was regarded as a toxic eruption of unknown origin. As more cases were observed it became evident that this was a definite disease entity which followed a course similar to the acute exanthemata of infancy and childhood.

Veeder and Hempleman [1] recently reported a series of cases of a similar nature. They believe that they are describing the same condition that was described by Zahorsky [2] in 1910. My series of cases corresponds almost exactly with those of Veeder and Hempleman and Zahorsky.

Age.—The disease occurred in patients whose ages ranged from 9 months to 2 years. None of the infants were breast fed. Veeder and Hempleman have observed this condition in older children, but state that it has a predilection for infants.

Symptoms.—In all cases the illness occurred suddenly, the patients being perfectly well the day before. At the onset the prominent symptoms were fever, irritability, and refusal of food. The fever during the pre-eruptive period ranged from 102 to 105 F. The drop in temperature and the appearance of the eruption were always coincident with a lessening of irritability and a return of the appetite. The extensive eruption apparently caused no symptoms. In spite of the high fever the infants were not toxic.

Physical Signs.—These were conspicious by their absence. No cause for the temperature could be found before the eruption appeared. Careful routine examination including the ears was always negative.

Eruption.—The lesions were mostly macules, but papules were also present. They were about one-eighth of an inch in diameter and pale or rose red in color. They could be pressed out easily and no hemorrhagic lesions were ever seen. The eruption had the general blotchy appearance of a measles eruption, but no crescentic arrangement was ever seen. The eruption usually was noticed first on the neck and quickly spread downward. It was more pronounced on the neck and trunk than on the face and extremities. The entire course of the eruption was from two to three days. There was a very faint desquamation.

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1. Veeder and Hempleman: A Febrile Exanthem Occurring in Childhood, Tr. Am. Ped. Soc., June, 1921; abstr. J. A. M. A. **77**:312, 1921.

2. Zahorsky: Roseola Infantum, Pediatrics, **22**:60 (July 23) 1910.

Through the courtesy of Drs. Veeder and Hempleman, I was able to obtain a copy of their paper before publication and also to obtain the reference to Zahorsky's article.

Laboratory Findings.—The urine was examined in all cases and was found to be uniformly negative. Veeder and Hempleman call particular attention to the blood picture which is characterized by a leukopenia and a high lymphocytosis. In three of my cases in which a blood count was done, exactly the same picture was found. The lymphocytosis in the three cases was always over 80 per cent. This seems to be an important point.

Contagion.—The disease occurred in only one member of each family. It did not seem to be contagious.

Diagnosis.—When the temperature drops and the eruption appears, the diagnosis is very easy. During the pre-eruptive period one thinks of pyelitis, otitis media and pneumonia. The blood count and differential picture is very helpful at this stage. The eruption resembles measles more than anything else. However, measles is easily excluded by the absence of Koplik's spots and by the fact that as the eruption appears, the temperature drops and the patient seems perfectly well.

Rubella is also to be considered. In rubella there is usually no prodromal period of fever before the eruption such as was observed in these cases. In rubella the fever is much lower than is observed here. Also there is no post-auricular adenitis as is usually seen in rubella.

The only exanthem which has a stormy pre-eruptive period, followed by well being when the eruption appears, is variola. However, the condition has nothing in common with variola. There also remains to be considered toxic or anaphylactic eruptions caused by various proteins, food and bacterial. No definite evidence of this etiology could be demonstrated. None of the patients were taking any drugs before their illness.

Treatment.—The treatment is purely symptomatic. The eruption disappears spontaneously. There have been no complications.

DISCUSSION

It is my belief that this is a definite disease entity, not described in any of the present day pediatric textbooks. Veeder and Hempleman have proposed the name "Exanthem Subitum" to describe this condition as the most characteristic thing is the sudden appearance of the eruption on the fourth day.

CONCLUSIONS

An unusual disease occurring in infants is described. It is characterized by a pre-eruptive period of three to four days with high fever. On the fourth day there is a sudden drop in temperature coincident with an eruption. The eruption last one to two days and is maculopapular resembling measles. The etiology is unknown.

THE RESPIRATORY EXCHANGE IN A CASE OF BILIARY ATRESIA*

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The study of the respiratory exchange in a case of biliary atresia was undertaken to see whether the presence of large quantities of unabsorbed fat in the intestine had any influence on the absorption of carbohydrate. It is held by many that fat is the fons et origo mali of many cases of marasmus and malnutrition in infancy. It has been shown that in these conditions fat can be absorbed from the intestine [1] and metabolized in the tissues,[2] but it has not been shown whether the presence of fat in the intestine is any impediment to the absorption of other forms of food. This case of biliary atresia seemed to afford an opportunity of seeing whether large quantities of unabsorbed fat in the intestine inhibited the absorption of carbohydrate.

REPORT OF CASE

History.—J. S., female, aged 8 weeks, was admitted to the Royal Hospital for Sick Children, Glasgow, Jan. 10, 1921, with a history of jaundice of two weeks' duration. She was a first child and the parents were healthy. Labor occurred at full term; it was normal and the child seemed to be healthy at birth. It throve well at first. At the age of 6 weeks jaundice was first noticed, and this gradually increased in intensity. The stools which at first were pigmented are said gradually to have become paler and paler. Otherwise the child seemed healthy and was increasing in weight.

Physical Examination.—On admission the child appeared to be fairly well nourished, weight 7 pounds 9 ounces, but deeply jaundiced. The examination of the heart and lungs was negative. The liver was palpable two finger breadths below the costal margin. The spleen was not palpable. The stools were clay colored and did not contain hydrobilirubin. The urine contained much bile pigment. The von Pirquet and Wassermann reactions were negative. From January till April 27 the child progressed slowly. The weight increased to 9 pounds 10 ounces, but the jaundice gradually increased in intensity. On April 27, the temperature rose. An ulcerative pharyngitis developed. The blood culture gave a pure growth of streptococci. The child died, May 4.

Necropsy Report.—At the postmortem examination no traces of the gallbladder or bile ducts could be found, their position being occupied by strands of fibrous tissue without any evidence of a lumen. The liver showed a generalized cirrhosis of the biliary type. The pancreatic ducts opened normally in the papilla of Vater. Otherwise, the findings were those of a general septicemia.

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* Received for publication, Aug. 25, 1921.
* From the Royal Hospital for Sick Children.
* The expenses of this investigation were defrayed by the Medical Research Council.

1. Hutchison: Quart. J. M. **13**:277 (April) 1920.
2. Fleming: Quart. J. M. **14**:171 (Jan.) 1921.

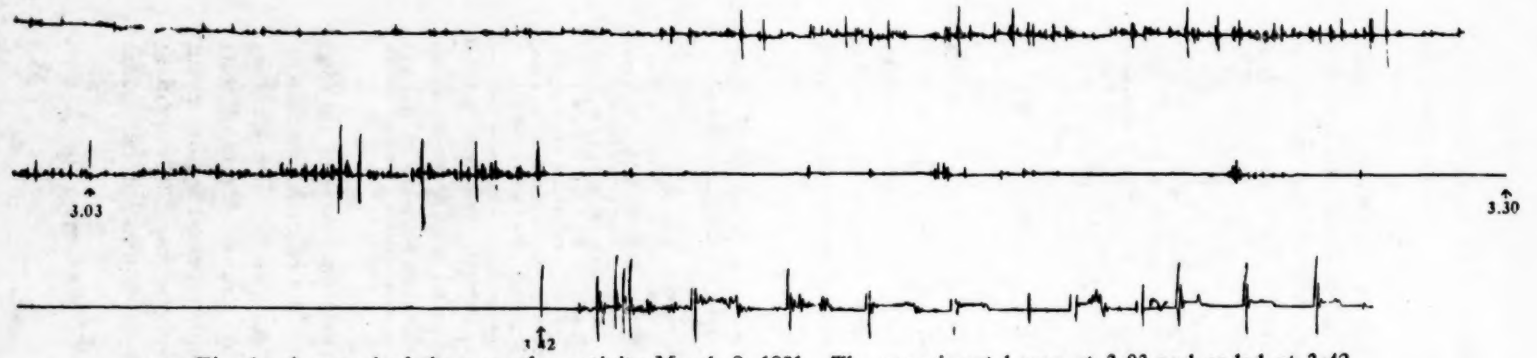

Fig. 1.—A record of the muscular activity March 8, 1921. The experiment began at 3:03 and ended at 3:42.

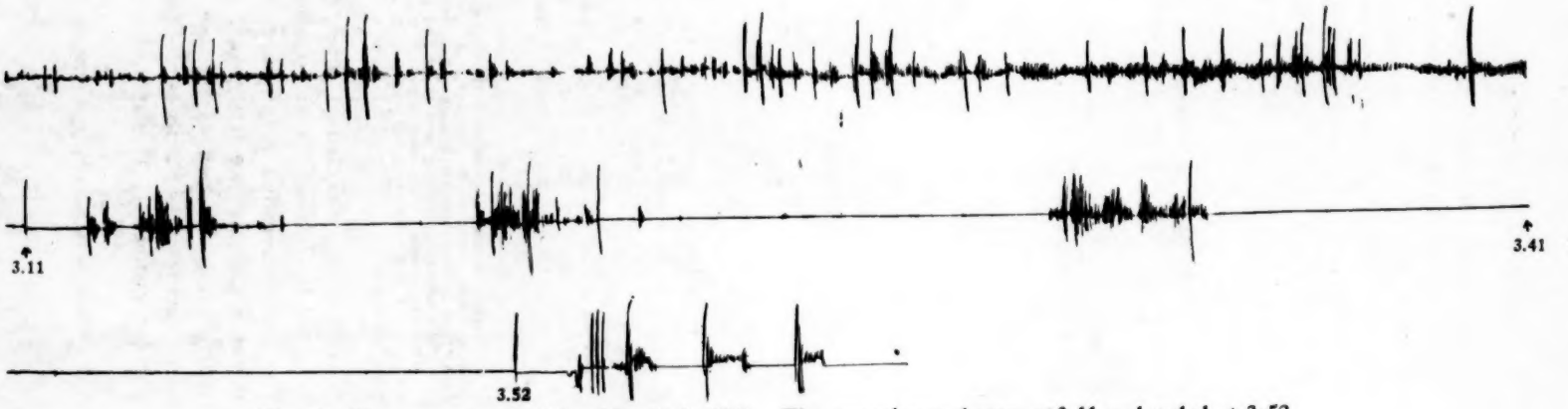

Fig. 2.—The muscular activity March 9, 1921. The experiment began at3:11 and ended at 3:52.

During the residence in hospital various milk mixtures were tried. It was found that large quantities of carbohydrate were necessary for the child to increase in weight, while large quantities of fat (over 5 per cent.) even with the retention in the diet of the full dose of carbohydrate, caused a loss of weight. The fat in the stools was estimated when the child was on a poor fat diet, a diet with normal amounts of fat in it, and a rich fat diet. I am indebted to Dr. Telfer for the stool examination (table 1).

TABLE 1.—PERCENTAGES OF FAT IN THE DRIED STOOLS

	Period 1 0.8% Fat	Period 2 3.0% Fat	Period 3 5.3% Fat
Neutral fat	10.68	5.50	15.26
Free fatty acid (as oleic acid)	25.34	39.29	48.43
Combined fatty acids	28.44	33.23	17.17
Total	64.46	78.02	80.86

These figures were arrived at by collecting the feces for several days of each period. Unfortunately, the total excretion of fat was not estimated and compared to the total ingested. The normal amount of fat in the infant's stool is about 30 per cent. So it is clear that in this case, even when on a fat poor diet, there was an enormous excess of fat in the bowel.

TABLE 2.—ESTIMATION OF THE RESPIRATORY EXCHANGE ON VARIOUS FAT DIETS

Date	Liter O_2 per Hour	Liter CO_2 per Hour	Respiratory Quotient	Calories per 24 Hours	Activity*	Weight in Kilo	Diet†
3/ 8/21	2.83	2.88	1.01	343	III	3.85	0.8% fat, 100 c.c.; Sl., 4 gm.;
3/ 9/21	2.84	2.87	1.01	344	III	3.91	Sl., 4 gm.; 8 times
3/11/21	2.86	2.80	0.97	344	III	3.97	3% fat, 100 c.c.; S., 4 gm;
3/15/21	3.36	3.40	1.01	407	IV	4.08	Sl., 4 gm.; 8 times
3/17/21	2.76	2.25	0.81	319	I	3.96	5.3% fat, 100 c.c.; 8 times
3/18/21	3.21	2.96	0.92	380	IV	3.91	5.3% fat, 100 c.c.; S., 4 gm.; Sl., 4 gm.; 8 times

* Varying degrees of muscular activity are indicated by numbers. The lowest degree of activity being indicated by the numeral I and the highest by the numeral VI. With an activity of I the subject is usually asleep and the movements are minimal, with an activity of VI the subject is moving constantly and probably crying.

† 1% fat = cow's milk containing 1% fat; 3% fat = cow's milk containing 3% fat; 5.3% fat = cow's milk containing 5.3% fat; S. = cane sugar; Sl. = a mixture of starch dextrin and cane sugar.

The infant was fed at three hour intervals and was on the diet indicated in Table 2 for at least twelve hours before each experiment. The Benedict-Talbot respiratory apparatus was used.[3] The accuracy of the apparatus was frequently verified by means of control tests with alcohol. In each experiment a preliminary period lasting about thirty minutes was put through. In this period the respiratory quotient was not estimated. It was followed by the experiment proper which

3. Benedict and Talbot: Carnegie Institution of Washington, D. C., 1914.

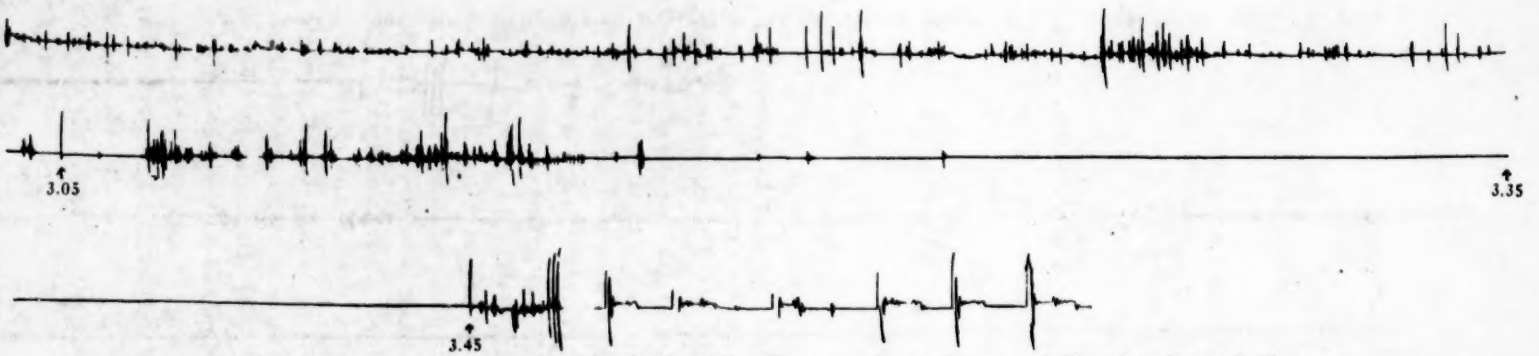

Fig. 3.—The muscular activity March 11, 1921. The experiment began at 3:05 and ended at 3:45.

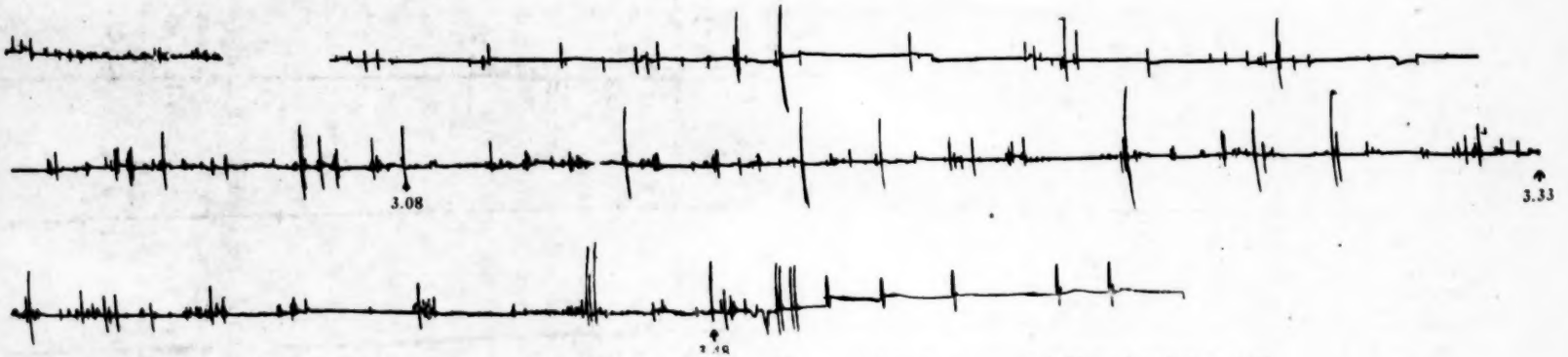

Fig. 4.—The muscular activity March 15, 1921. The experiment began at 3:08 and ended at 3:48.

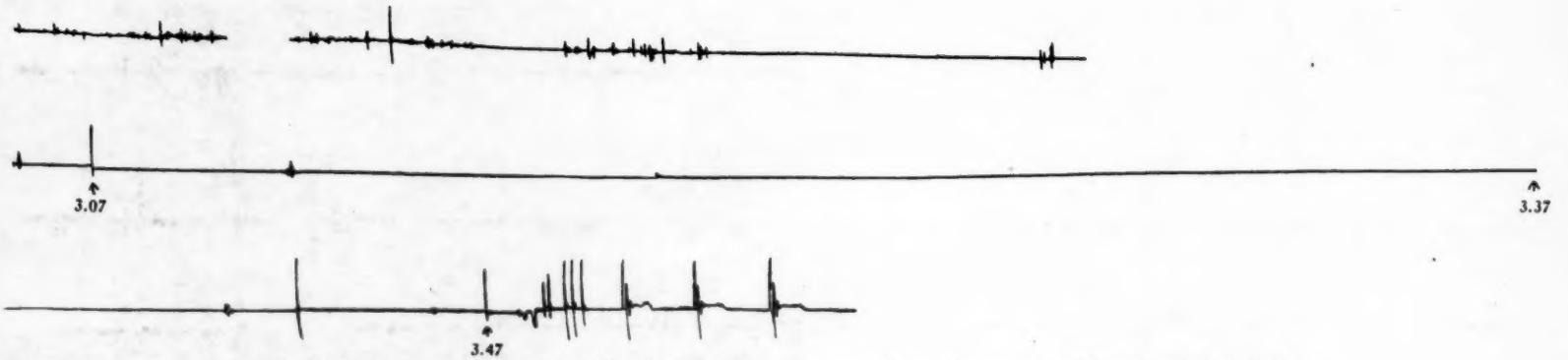

Fig. 5.—The muscular activity March 17, 1921. The experiment began at 3:07 and ended at 3:47.

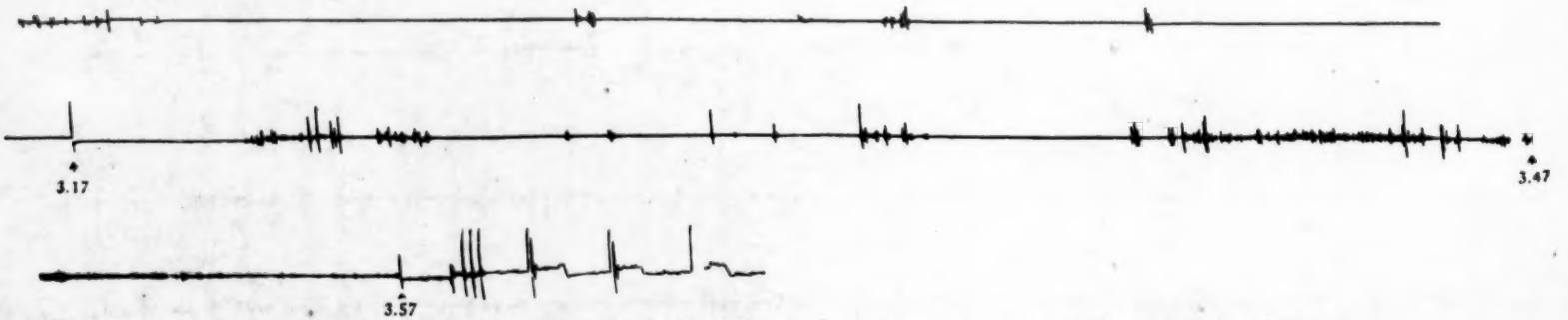

Fig. 6.—The muscular activity March 18, 1921. The experiment began at 3:17 and ended at 3:57.

lasted for from thirty-nine to forty-one minutes. The child was fed from forty-two to fifty-three minutes before the commencement of the experiments to determine the respiratory quotient. Figures 1 to 6 give a graphic record of the muscular activity of the child during each of the experiments. Fortunately almost identical conditions of muscular activity were obtained on March 8, 9 and 11, and also on March 15 and 18.

In the first three experiments in which the activity was the same, the oxygen intake and the carbon dioxide excretion were remarkably constant, and the respiratory quotients also were almost the same, in spite of the fact that in the third experiment three times as much fat was given as in the first two. In the fourth experiment, although the activity was greater the respiratory quotient remained at 1.01. On this day the diet was the same as that on March 11. On March 17 most of the carbohydrate had been cut out of the diet and the fat had been increased to 5.3 per cent. The respiratory quotient fell to 0.81. On March 18 the rich fat diet was maintained but the carbohydrate was brought up to the same strength as was given in the first four experiments. The respiratory quotient on this occasion was 0.92.

Although theoretically a respiratory quotient of 1.0 means that carbohydrate alone is being metabolized, in practice this is not necessarily the case for protein metabolism must always be taking place and this would tend to reduce the respiratory quotient. Probably a respiratory quotient in the neighborhood of 1.0 means that metabolism is being carried on largely at the expense of carbohydrate and that at the same time some carbohydrate is being converted into fat.

In the first four experiments, when moderate amounts of fat were being given (up to 3 per cent.), this is probably what was taking place and these moderate quantities of fat in the diet were no hindrance to the absorption of carbohydrate. In the fifth experiment there is a sudden fall in the respiratory quotient. This may be accounted for in two ways. It will be noted that much more fat and much less carbohydrate were given on that day and the respiratory quotient of 0.81 may be explained either by the diminished quantity of carbohydrate available for combustion or by the excessive quantities of fat causing delay in the absorption of the diminished amount of carbohydrate in the diet. Probably both these factors played their part in the last experiment when the same quantity of fat was given as on the previous day but the amount of carbohydrate in the food was brought up to the same level as in the first experiment. On this occasion the respiratory quotient lay midway between that obtained on the fifth day and on the first 4 days. Probably, therefore, large quantities of fat delayed the absorption of carbohydrate in this case to a moderate degree.

BACTERIOLOGIC STUDIES OF ONE HUNDRED AND SIXTY-FIVE CASES OF PNEUMONIA AND POSTPNEUMONIC EMPYEMA IN INFANTS AND CHILDREN*

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INTRODUCTORY

Extensive bacteriologic studies of pneumonias among adults have established certain cardinal facts.

1. Lobar pneumonia is caused by the pneumococcus—either by one of three fixed biologic types, designated arbitrarily as Types I, II and III, or by a member of the heterogeneous group, called Group IV, which embraces all other strains.[1] Under Type II a series of fixed subtypes has been shown, all closely related to the master strain; these are called atypical Type II.[2]

2. Under nonepidemic conditions the usual proportionate incidence of infections caused by the different types is about as follows: Type I, 33 per cent.; Type II, 25 per cent.; Type II, atypical, 7 per cent.; Type III, 12 per cent., and Group IV, 22 per cent.[3]

3. The average mortality of lobar pneumonia caused by pneumococcus Type I is from 25 to 30 per cent. (in cases not treated with serum); by Type II, from 25 to 30 per cent.; by Type II atypical, 25 per cent.; by Type III, from 45 to 50 per cent., and by members of Group IV, from 15 to 20 per cent.

4. The presence of the pneumococcus in the blood stream is of grave prognostic significance. Generally speaking, the mortality is in direct proportion to the severity of the bacteraemia. Invasion of the blood by pneumococcus Type III almost always forecasts a fatal outcome. The elimination of specific soluble substance (precipitin) by way of the kidney is also of prognostic value. The greater the amount of such substance in the urine the more grave the outlook.[1]

5. In the mouths of healthy individuals pneumococci of Group IV are common, and of Types II atypical and III not rare. Pneumococci of Types I and II are rarely found in normal individuals.[4] It follows

* Received for publication, Oct. 17, 1921.

* From the Pneumonia Service of the Boston City Hospital.

1. Avery, Chickering, Cole and Dochez: Monograph No. 7, Rockefeller Institute M. Research, Oct. 16, 1917.
2. Avery, O. T.: J. Exper. M. **21**:804, 1915.
3. Cole, R.: Nelson Loose-Leaf Med. **1**:203, 1920.
4. Stillman, E. G.: J. Exper. M. **24**:651, 1916; Ibid. **26**:513, 1917.

that in the examination of sputum from pneumonia patients the finding of Type I or Type II is most significant, of Type II atypical or Type III something less so, while the finding of a member of Group IV is dubiously interpretable as regards its etiological rôle in the disease.

6. Against pneumococcus Type I a potent therapeutic serum has been prepared which, early and efficiently employed, has notably reduced the mortality for that type.

For bronchopneumonia by very nature no such array of clean-cut facts can be mustered. Bronchopneumonia is not an entity; the term represents a group of pulmonary infections which vary considerably in pathologic detail according to the exciting agent and circumstances of origin. Of the many organisms which may stand in causal relationship to bronchopneumonia the most important are probably the pneumococci, the streptococci, *Staphylococcus aureus,* and the influenza bacillus. The relative frequency of these and other organisms cannot be given. Of the pneumococcus it may safely be said that the fixed types are much less commonly present than in lobar pneumonia. The hemolytic streptococcus is a particularly important organism. Its tendency to produce empyema or other infection of serous membranes and the very high mortality which follows in its train have united to give it a place of unenviable eminence.[5] Only numerically less important is *Staphylococcus aureus,* which when operative in bronchopneumonia nearly always gives rise to fatal results.[6]

Lobar pneumonia differs in its behavior in the child and in the adult. It is usually a much milder disease in the earlier period, and the death rate is materially lower. Hence, it is clear that the facts which have been established for the disease as it occurs in adult life cannot wholly apply in the case of children.

The relative mildness of lobar pneumonia in childhood may reasonably be explained by one of two hypotheses: (1) the bulk of cases are caused by the less virulent strains of pneumococci, such as are comprised in Group IV; or, (2) the incidence of fixed types being much as for adults, the child opposes to them a better natural resistance than does the average adult.

Definitely to determine which of these hypotheses is correct is a matter of some importance, certainly as regards the carrier problem, if not so much from the standpoint of individual prognosis and treatment. For if most of the pneumonias of childhood are caused by the

5. Cole and MacCallum: J. A. M. A. **70**:1146 (April 20) 1918. MacCallum, W. G.: Monograph No. 10, Rockefeller Institute M. Research, April 16, 1919. Brooks and Cecil: Tr. Assn. Am. Phys. **33**:83, 1918.

6. Chickering and Park: J. A. M. A. **72**:617 (March 1) 1919. Griffith, J. P. C.: Diseases of Infants and Children, Philadelphia **2**: 1920.

common mouth organisms, these small patients are of far less menace as propagators of disease than if they harbor the fixed, and for adults much more virulent, fixed types.

In short, the particular problem which offers itself for solution is to determine the degree of parallelism between the etiology of the pneumonias of childhood and that of the pneumonias of adult life. Such a comparison must be easier to initiate in the case of lobar pneumonia than in the case of lobular pneumonia, but the occurrence in children of the most dreaded organisms associated with adult broncho-pneumonia (such, for example, as the hemolytic streptococcus) would have a similar bearing on the carrier problem as would the occurrence of the fixed types of pneumococci.

The question of the parallelism of etiology in these two periods of life must still be considered as an open one. It is true that such few bacteriologic studies as have been reported for the pneumonias of infancy and childhood have led to the statement that the common mouth forms of pneumococci (Group IV) are largely responsible for these diseases during that period, that the fixed types are relatively infrequent, and specifically that Type I pneumococcus is comparatively rare. In reviewing the studies from which these statements are derived, however, one is struck with the numerical inadequacy of the data. Too few cases have been reported to admit of any general definite conclusions.

The studies which are reported in this paper are intended to furnish additional data along these lines. One hundred and sixty-five patients, 12 years of age or younger, ill with lobar pneumonia, broncho-pneumonia, or complications of these diseases, have been investigated. The results of these studies are presented in the spirit of suggestion rather than of finality, and with the hopes that further studies may be undertaken in a field that promises to be important.

MATERIALS AND METHODS

These studies are based on sputum examinations, blood cultures, cultures from pleural fluids, or from the lung itself, and in rare instances from spinal fluids. It has not been possible to attempt a study of the precipitin reaction in the urine.

Sputum.—There has been, on the whole, less difficulty in securing sputum for examination than might have been expected. The first method we employed was to introduce a sterile rubber catheter (No. 18 or 20 French) against the patient's pharyngeal wall, taking care to avoid the tongue, tonsils and buccal mucous membrane. The active gag reflex attendant on this procedure was usually productive of sufficient sputum to fill the eye of the catheter. This material was at once washed into 1 or 2 c.c. of sterile broth or salt solution, the

tube shaken to secure as even an emulsion as possible, and a sample introduced into the peritoneum of a white mouse. Any remaining material could be used for smear preparations. Subsequently it appeared that a medium-sized sterile cotton swab was quite as efficient as the catheter on which to catch the sputum, besides obviating the necessity of repeated sterilizations.

Cultures from the peritoneal exudate and heart's blood of the mouse have been handled in the usual manner.[1] In every instance the organism (or organisms) reported has been obtained by means of mouse passage; it has not been feasible to make direct plate cultures of the sputum as obtained.

Blood cultures have been made in suitable cases. Small superficial veins have constituted a real and frequent difficulty. To withdraw sufficient blood may require repeated attempts, and besides the attendant discomfort, much manipulation greatly increases the liability of contamination. In consequence we have met with some failures, and in other cases we have not even attempted the procedure, when, ceteris paribus, we should have wished these data. In other cases in which the disease has been obviously very mild or well advanced toward convalescence a blood culture seemed superfluous.

In infants we have occasionally resorted to puncture of the longitudinal sinus through the open fontanel. In marasmic and desiccated infants, however, we have sometimes feared to withdraw even the amount of blood necessary for culture. Hence the infrequency of this procedure.

The blood cultures were made in flasks, from 3 to 4 c.c. blood to 50 c.c. of a beef infusion broth, p_H, 7.8. Cultures were incubated at 37 C. for five or six days before being discarded as sterile. In the event of growth, the usual means were employed to identify the organism.

Lung Punctures.—Although in the pathologic sense no untoward effects are to be anticipated from the puncture of a solid lung, I am reluctant to tap the chest unless there is suspicion of fluid. At best, it is a somewhat painful procedure, and often excites in the child patient a terror which for several days to come reawakens with every appearance of the physician. In the event of a dry tap, when the needle has entered the lung, it has been our habit to wash out the tip in a tube of sterile broth. Thus not infrequently we have obtained a culture direct from the seat of the pathologic process. A few lung cultures have been made by this means directly after death.

Pleural Fluids.—No matter what the character of the fluid was, we have cultured it directly into broth. Further plate cultures from this parent broth culture have been made as indicated.

Spinal fluids were taken only from the few cases in which there was reasonable suspicion of meningeal involvement. Cultures were made into broth, on solid medium, or both.

BACTERIOLOGY

Ninety-eight cases of lobar pneumonia, fifty-two cases of bronchopneumonia, and fifteen cases of postpneumonic empyema have been studied. Those cases of lobar or of bronchopneumonia in which empyema developed under observation have been classified in this schedule under the name of original disease. It should be stated that in making the differential diagnosis between lobar and lobular pneumonia we have been guided solely by clinical and roentgenologic findings, and in no instance by the type of organism recovered from the case.

The bacteriologic findings in the above mentioned cases are fully shown in Table I, which gives the organism or organisms isolated in each, and the particular source of such findings, the age, principal complications, and result in each case.

TABLE 1.—Bacteriological Findings in One Hundred and Sixty-Five Cases of Pneumonia and Empyema in Infants and Children

Case No.	Age	Diagnosis	Organism	Sputum	Blood	Lung	Pleural Fluid	Result	Remarks
19	10	Lobar	Pneumococcus I	+	..	..	..	Rec.	
22	9	Lobar	Pneumococcus I	+	..	..	..	Rec.	
23	10	Labor	Pneumococcus I	+	..	..	..	Rec.	
65	4	Labor	Pneumococcus I	+	..	..	..	Rec.	
131	1 5/12	Lobar	Pneumococcus I	+	..	..	..	Rec.	
116	5	Lobar	Pneumococcus I	+	S.*	..	..	Rec.	
101	1 2/12	Lobar	Pneumococcus I	+	S.	..	..	Rec.	
98	11	Lobar	Pneumococcus I	+	S.	..	..	Rec.	Urine precipitin +
72	8	Lobar	Pneumococcus I	+	..	+	..	Rec.	Serum treatment
73	9	Lobar	Pneumococcus I	+	..	+	..	Rec.	Serum treatment
45	7	Lobar	Pneumococcus I	..	..	+	..	Rec.	Serum treatment
68	7	Lobar	Pneumococcus I	..	+	..	..	Died	Laryngeal diphtheria complicating..
78	5	Lobar	Pneumococcus I	..	+	..	..	Rec.	
80	11	Lobar	Pneumococcus I	..	+	..	..	Rec.	
75	6	Lobar	Pneumococcus I	..	..	..	+	Rec.	Empyema; operation
77	12	Lobar	Pneumococcus I	..	..	..	+	Rec.	Empyema; operation
79	2 4/12	Lobar	Pneumococcus I	..	..	..	+	Rec.	Empyema; operation
129	10/12	Lobar	Pneumococcus I	..	..	..	+	Died	Empyema; operation
74	6	Lobar	Pneumococcus I	..	+	+	+	Rec.	Empyema; operation
69	4 6/12	Lobar	Pneumococcus I	..	..	..	+	Died	Empyema; operation
123	7	Lobar	Pneumococcus I	..	+	+	+	....	Empyema; operation
112	5	Lobar	Pneumococcus I	..	..	+	+	Rec.	Empyema; operation
107	4	Lobar	Pneumococcus I	—	+	+	+	Rec.	Empyema; operation
			Pneumococcus IV	+	—	—	—		
71	7	Lobar	Pneumococcus I	—	..	..	+	Rec.	Empyema; operation
			Pneumococcus IV	+	..	..	—		
115	11	Lobar	Pneumococcus I	—	S.	..	+	....	Empyema; operation
			Strep. viridans	+	..	..	—		
20	6	Lobar	Pneumococcus I	+	..	..	..	Rec.	
21	9	Lobar	Pneumococcus I	+	+	..	S.	Rec.	
			Pfeiffer	+					
110	7	Lobar	Pneumococcus I	+	S.	..	+	Rec.	Spinal fluid sterile; pneumothorax; diphtheria; scarlet fever; operation
			Pfeiffer	+	..	..	—		
54	6	Lobar	Pneumococcus I	+	S.	..	..	Rec.	

* Sterile. † Contaminated. ‡ Reinfection, three weeks later. §Mouse lived.

TABLE 1.—Bacteriological Findings in One Hundred and Sixty-Five Cases of Pneumonia and Empyema in Infants and Children.—*(Continued)*

Case No.	Age	Diagnosis	Organism	Sputum	Blood	Lung	Pleural Fluid	Result	Remarks
3	10	Lobar	Pneumococcus II	+	S.	S.	..	Rec.	
119	3	Lobar	Pneumococcus II	+	..	..	..	Rec.	
11	8	Lobar	Pneumococcus IIa	+	..	..	..	Rec.	
70	10	Lobar	Pneumococcus III	+	†	..	..	Rec.	
105	6	Lobar	Pneumococcus III	+	S.	..	..	Rec.	
1	9	Lobar	Pneumococcus III Pfeiffer	+ +	..	S.	..	Rec.	
50	9	Lobar	Pneumococcus III Pfeiffer	+ +	S.	..	..	Rec.	
51	12	Lobar	Pneumococcus III Pfeiffer	+ +	S.	..	..	Rec.	
5	12	Lobar	Pneumococcus III Pfeiffer	+ +	S.	..	..	Rec.	
18	12	Lobar	Pneumococcus III Strep. viridans	+ +	..	..	..	Rec.	
7	5	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
9	6	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
10	8	Lobar	Pneumococcus IV	+	..	..	S.	Rec.	
12	7	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
13	9	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
15	12	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
16	6	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
56	2 10/12	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
57	1 5/12	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
59	8/12	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
96	6	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
97	12	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
62	2	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
64	6	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
99	2	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
99‡	..	Lobar	Pneumococcus IV	+					
100	4	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
103	12	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
104	6	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
106	5	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
109	4	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
111	7	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
113	11	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
120	3	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
121	3	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
124	1 5/12	Lobar	Pneumococcus IV	+	..	..	..	Rec.	
127	2 6/12	Lobar	Pneumococcus IV	+	S.	..	..	Rec.	
126	9	Lobar	Pneumococcus IV	+	S.	S.	..	Rec.	
67	1 8/12	Lobar	Pneumococcus IV	..	..	+	..	Rec.	
76	7	Lobar	Pneumococcus IV	..	..	..	+	Rec.	Empyema; operation
66	8	Lobar	Pneumococcus IV	..	..	+	..	Died	Lung puncture postmortem
130	1 3/12	Lobar	Pneumococcus IV	..	†	+	..	Died	Lung culture postmortem
4	2	Lobar	Pneumococcus IV Pfeiffer	+ +	†	..	..	Rec.	
6	6 6/12	Lobar	Pneumococcus IV Pfeiffer	+ +	..	..	..	Rec.	
14	12	Lobar	Pneumococcus IV Pfeiffer	+ +	..	..	..	Rec.	
24	9	Lobar	Pneumococcus IV Pfeiffer	+ +	..	..	..	Rec.	
60	2	Lobar	Pneumococcus IV Pfeiffer	+ +	..	..	..	Rec.	
118	4	Lobar	Pneucoccoccus IV Pfeiffer	+ +	S. ..	+ +	..	Rec.	
102	7	Lobar	Strep. hemolyticus	§	S.	+	+	Died	Empyema; operation; general peritonitis
48	10	Lobar	Strep. hemolyticus Strep. viridans Pfeiffer	+ + +	S.	..	..	Rec.	
17	6	Lobar	Strep. viridans	+	..	..	..	Died	Laryngeal diphtheria complicating
2	7	Lobar	Strep. viridans	+	..	..	..	Rec.	
25	3	Lobar	Strep. viridans	+	..	..	..	Rec.	
8	11	Lobar	Strep. viridans	+	..	..	..	Rec.	
49	3	Lobar	Strep. viridans Pfeiffer	+ +	..	..	..	Rec.	

TABLE 1.—BACTERIOLOGICAL FINDINGS IN ONE HUNDRED AND SIXTY-FIVE CASES OF PNEUMONIA AND EMPYEMA IN INFANTS AND CHILDREN.—*(Continued)*

Case No.	Age	Diagnosis	Organism	Sputum	Blood	Lung	Pleural Fluid	Result	Remarks
135	7	Lobar	Unknown	..	..	S.	..	Rec.	
136	1 5/12	Lobar	Unknown	..	..	S.	..	Rec.	
137	11	Lobar	Unknown	..	..	S.	..	Rec.	
138	10	Lobar	Unknown	..	..	S.	..	Rec.	
139	7	Lobar	Unknown	..	..	S.	..	Rec.	
140	8	Lobar	Unknown	..	S.	..	..	Rec.	
141	6	Lobar	Unknown	S	..	S.	..	Rec.	
142	3	Lobar	Unknown	..	..	S.	..	Rec.	
143	1 7/12	Lobar	Unknown	..	S.	..	..	Rec.	
144	12	Lobar	Unknown	S	..	..	..	Rec.	
145	12	Lobar	Unknown	S	..	..	..	Rec.	
146	8	Lobar	Unknown	..	+	..	S.	Rec.	
147	9	Lobar	Unknown	S	S.	..	..	Rec.	
148	3	Lobar	Unknown	S	S.	..	..	Rec.	
133	10	Lobar	Unknown	S	S.	..	..	Rec.	
33	8	Broncho	Pneumococcus I	+	..	S.	..	Rec.	
36	9	Broncho	Pneumococcus I	+	..	..	..	Rec.	
95	2 6/12	Broncho	Pneumococcus I	..	+	..	+	Died	
			Pfeiffer	..	—	..	+		
84	7	Broncho	Pneumococcus II	+	—	S.	..	Died	
			Strep. hemolyticus	—	+				
42	9/12	Broncho	Pneumococcus IIa	+	0	S.	..	Died	Lung puncture postmortem
30	1 3/12	Broncho	Pneumococcus III	+	..	..	..	Rec.	
35	8	Broncho	Pneumococcus III	+	..	..	..	Rec.	
			Pfeiffer	+					
58	1 4/12	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
29	1 11/12	Broncho	Pneumococcus IV	+	..	..	..	Died	Spinal fluid +; pneumococcus meningitis complicating
117	3	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
82	8/12	Broncho	Pneumococcus IV	..	..	+	..	Died	Lung puncture postmortem
94	3	Broncho	Pneumococcus IV	..	..	+	..	Died	Lung puncture postmortem
91	2	Broncho	Pneumococcus IV	..	..	..	+	Died	Empyema; operation
47	1	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
122	2	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
134	3	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
28	7/12	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
			Pfeiffer	+					
31	4	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
			Pfeiffer	+					
32	1 6/12	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
			Pfeiffer	+					
34	2	Broncho	Pneumococcus IV	+	..	..	..	Died	
			Pfeiffer	+					
55	11	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
			Pfeiffer	+					
38	2	Broncho	Pneumococcus IV	+	..	..	..	Imp.	
			Pfeiffer	+					
26	1 2/12	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
			Strep. hemolyticus	+					
			Pfeiffer	+					
46	8	Broncho	Pneumococcus IV	+	..	..	..	Rec.	
			Strep. hemolyticus	+					
			Pfeiffer	+					
85	1 6/12	Broncho	Pneumococcus IV	..	..	+	..	Died	
			Staph. aureus	..	..	+			
90	1 4/12	Broncho	Pneumococcus IV	..	..	+	..	Died	
			Strep. hemolyticus	..	..	+			
61	8/12	Broncho	Strep. hemolyticus	+	..	..	..	Died	
92	1	Broncho	Sterp. hemolyticus	..	..	..	+	Died	Empyema; operation
93	11	Broncho	Strep. hemolyticus	..	..	..	+	Died	Empyema; operation
40	9/12	Broncho	Strep. hemolyticus	+	..	..	..	Died	
			Pfeiffer	+					
41	6/12	Broncho	Strep. hemolyticus	+	..	..	..	Died	
			Pfeiffer	+					
27	1 5/12	Broncho	Strep. hemolyticus	+	..	..	..	Died	
			Strep. viridans	+					
86	7	Broncho	Strep. hemolyticus	—	..	..	+	Rec.	Empyema; operation
			Strep. viridans	—	..	..	+		
			Staph. aureus	+	..	..	+		
			Yeast	+	..	..	+		

TABLE 1.—Bacteriological Findings in One Hundred and Sixty-Five Cases of Pneumonia and Empyema in Infants and Children—(*Continued*)

Case No.	Age	Diagnosis	Organism	Sputum	Blood	Lung	Pleural Fluid	Result	Remarks
39	6	Broncho	Strep. viridans	+	..	..	..	Rec.	
37	2 6/12	Broncho	Strep. viridans Pfeiffer	+ +	..	..	..	Died	
83	8	Broncho	Staph. aureus	..	+	+	..	Lived	Multiple abscess
89	4	Broncho	Staph. aureus	+	..	+	..	Died	
44	1 6/12	Broncho	Staph. aureus	..	..	+	..	Died	
81	4 6/12	Broncho	Staph. aureus Pneumococcus IV	+ +	..	+ —	..	Died	
43	1 5/12	Broncho	Staph. aureus Strep. viridans	+ +	..	..	..	Died	
88	1	Broncho	Staph. albus Pfeiffer	.. +	..	+	..	Died	
128	1 6/12	Broncho	Unknown	..	S.	S.	..	Died	
132	4	Broncho	Unknown	§	S.	..	..	Rec.	
149	10	Broncho	Unknown	..	S.	..	..	Rec.	
150	1 3/12	Broncho	Unknown	..	S.	..	..	Rec.	
151	2	Broncho	Unknown	..	..	S.	..	Died	
152	2	Broncho	Unknown	..	..	†	..	Died	
153	7/12	Broncho	Unknown	..	..	..	..	Died	Spinal fluid sterile
154	2 6/12	Broncho	Unknown	..	S.	..	..	Died	
155	2/12	Broncho	Unknown	..	S.	..	..	Died	
156	2 6/12	Broncho	Unknown	..	S.	..	..	Died	
157	5	Broncho	Unknown	..	..	S.	..	Rec.	
158	3	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
159	11	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
160	2	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
161	9	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
162	10	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
163	8	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
164	4	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
165	5	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Died	Operation
166	2	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Died	Operation
167	7	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
168	9	Postpneumonic empyema	Pneumococcus I	..	..	..	+	Rec.	Operation
169	3	Postpneumonic empyema	Pneumococcus untyped	..	..	..	+	Rec.	Operation
170	3	Postpneumonic empyema	Strep. hemolyticus	..	..	..	+	Rec.	Operation
171	6	Postpneumonic empyema	Pneumococcus IV	..	..	..	+	Rec.	Operation
172	4	Postpneumonic empyema	Staph. aureus	..	..	..	+	Rec.	Operation

LOBAR PNEUMONIA

Sputum Examinations.—Seventy-three examinations were made. The findings of pneumococcus types in these sputums is shown in Table 2, arranged according to age groups, and indicating the mortality for each type within the age group.

TABLE 2.—PNEUMOCOCCUS TYPES AND MORTALITY IN SPUTUM IN SEVENTY-THREE CASES OF LOBAR PNEUMONIA IN INFANTS AND CHILDREN

Organism	First 2 Years			2 to 5 Years			6 to 12 Years			Totals		
		Percentage			Percentage			Percentage			Percentage	
	No.	Group	Mortality	No.	Group	Mortality	No.	Group	Mortality	No.	Total	Mortality
Pneumococcus I...	2	22.2	0	1	6.25	0	11	20.9	0	14	19.1	0
Pneumococcus II..	0	0	0	1	6.25	0	1	2.0	0	2	2.7	0
Pneumococcus IIa	0	0	0	0	0	0	1	2.0	0	1	1.4	0
Pneumococcus III	0	0	0	0	0	0	7	14.5	0	7	9.6	0
Pneumococcus IV	7	77.8	14.1	10	62.5	0	18	37.5	0	35	48.0	2.8
Other than pneumococcus.....	0	0	0	2	12.5	0	5	10 1	20	7	9.6	14 0
No result.....	0	0	0	1	6.25	0	6	12.0	0	7	9.6	0
Totals.....	9	0	11.0	16	100.0	0	48	100.0	2	73	100 0	2.7

In attempting to interpret these results we are confronted with the difficulties elsewhere mentioned. The finding in the sputum alone of a Group IV pneumococcus is always shadowed by the doubt as to whether it is really the pathogenic agent in the given case. One may sometimes doubt the significance of an atypical Type II or Type III pneumococcus so recovered. But the finding of a Type I or Type II is nearly always significant. The greater the number of Group IV's isolated, the more difficult are the results of estimation. In any series the error will almost invariably be on the side of understatement of the occurrence of fixed types rather than the reverse. The difficulty is, of course, to determine the degree of error. In this series we have been able actually to prove error in the sputum findings in only three cases; in these, blood, lung or pleural fluid showed the presence of a type pneumococcus which we had failed to isolate from the sputum. In fifty-seven cases, however, the only positive bacteriologic evidence was derived from the sputum. Hence, in passing any sort of judgment on the general dependability of our results our sole criterion is in the nature of indirect evidence, namely, as to how the findings in these children compare with the findings in a parallel series of cases in adults.

As regards the finding of other organisms than pneumococcus in an occasional sputum, I am not aware of any evidence to warrant the belief that such are ever the cause of lobar pneumonia. It is worthy of comment that Pfeiffer's bacillus is commonly present in the sputum of these children with lobar pneumonia. It has been found fourteen times, in association with pneumococcus, in this series of seventy-three sputum examinations.

Blood Cultures.—Forty-three blood cultures were made, representing 43.8 per cent. of the total cases studied. Six, or about 14 per cent., of these forty-three cultures were positive. Of the remainder four were contaminated; the others were sterile. All the positive cultures were of pneumococcus Type I. Only one of these patients died; in this case a laryngeal diphtheria complicated the pneumonia. Of the thirty-seven cases in which the blood was negative, two patients died; one of these had an uncomplicated pneumonia; the other died from empyema and general peritonitis which followed the pneumonia.

Lung punctures were performed in twenty cases (20.3 per cent. of the cases studied). With two exceptions these punctures were done during life. In ten cases the cultures were sterile. Of the 50 per cent. positive, six showed a pure culture of pneumococcus Type I, three a pure culture of pneumococcus Group IV, and one a mixed growth of pneumococcus Group IV and Pfeiffer's bacillus.

Pleural Fluids.—Fluid was found in the pleura in sixteen cases, or 16.3 per cent. of those studied. In three cases (3 per cent.) this fluid was sterile. The remaining thirteen cases (13.3 per cent.) represent frank empyema. Of the thirteen empyemas, eleven were caused by pneumococcus Type I, one by pneumococcus Group IV, and one by *Streptococcus haemolyticus* (Case 102).

Experience has shown that mixed pneumococcus infections are almost unknown. One, and only one, type is operative in a given individual at a given time. On this basis it has seemed only reasonable to attribute to the type pneumococcus recovered from the pleural pus the etiologic rôle in the antecedent pneumonia, though no supporting data were available, or in particular, even when the sputum finding was in contradiction.

On the other hand, it is well known that infection with *Streptococcus haemolyticus* is not uncommonly superimposed on pneumococcus pneumonia,[7] and, though proof is wanting, I am inclined to believe that such was the course of events in the one case which showed a streptococcus empyema.

Further observations on the bacteriology of empyema are reserved for a later portion of the paper.

BRONCHOPNEUMONIA

Sputum Examinations.—These were made in thirty-one of the fifty-two cases studied. The sputum bacteriology of these thirty-one cases is given in Table 3.

7. Clendenning, L.: Am. J. M. Sc. **156**:575, 1918.

TABLE 3.—Sputum Bacteriology in Thirty-One Cases of Bronchopneumonia in Infants and Children

	Cases	Deaths	Percentage Incidence	Percentage Mortality
Pneumococcus Type I	3	1	9.7	33.3
Pneumococcus Type II	1	1	3.2	100.0
Pneumococcus Type II atypical	1	1	3.2	100.0
Pneumococcus Type III	2	0	6.4	0.0
Pneumococcus Group IV	12	2	38.7	16.7
Streptococcus hemolyticus	4	4	12.9	100.0
Streptococcus viridans	2	1	6.4	50.0
Staphylococcus aureus	2	1	6.4	50.0
B. influenzae (e other organism)	11	..	35.4	
Other mixed bacteriology	4	1	12.9	25.0
Totals	31	12	....	38.7

It is known that any and all of these organisms may stand in etiologic relationship to bronchopneumonia. In three cases only is there confirmatory evidence actually to establish such relationship. The status of the other findings must in the last analysis remain conjectural, and unfortunately we have no standard with which to compare them. It is noteworthy that while the percentage figures show a decrease from lobar pneumonia in the total occurrence of fixed types, the reduction is largely at the expense of Type I, and that the increase in the occurrence of Group IV is relative rather than absolute. Other organisms than pneumococcus appear prominently in the list.

Blood Cultures.—Eleven cultures were made, representing about 21 per cent. of the cases studied. Of these three, or 27.2 per cent. were positive, the remainder were sterile. All the positive cultures showed pure growths—one of pneumococcus Type I, one of *Staphylococcus aureus,* and one of *Streptococcus haemolyticus.* From the last mentioned case, a Type II pneumococcus had been isolated from the sputum, and possibly the streptococcus represents a superimposed infection. The patient with pneumococcus septicemia also developed empyema. Both these patients died. The child with staphylococcus septicemia apparently recovered from the immediate infection, but has since become a confirmed invalid, suffering from multiple osteomyelitis.

Lung punctures were performed in eighteen, or 34.6 per cent. of the fifty-two cases. Positive cultures were obtained in nine (50 per cent.). The findings in these cases were as follows: pneumococcus Group IV, 2 cases; *Staphylococcus aureus,* 4 cases; pneumococcus, Group IV, plus *Staphylococcus aureus,* 1 case; pneumococcus Group IV, plus *Streptococcus haemolyticus,* 1 case; *Staphylococcus albus,* plus *B. Influenzae,* 1 case. Two of these punctures were performed postmortem. The only child among the entire series who lived was that from whom *Staphylococcus aureus* was obtained in pure culture—the same child mentioned above in whose blood this organism was present. There were only three recoveries among the nine cases in which lung punc-

tures were negative. There is little, then, to suggest that negative results in lung punctures warrant a more favorable prognosis.

POSTPNEUMONIC EMPYEMA

Fifteen cases admitted to the wards have been studied. While a large majority probably followed lobar pneumonia, the type of antecedent infection remains in some doubt, and I have preferred to classify the cases under the general heading "postpneumonic." In each of these cases a pure culture was obtained from the pleural pus. Of the fifteen cases, eleven showed pneumococcus Type I, one showed *Streptococcus haemolyticus,* one pneumococcus Group IV, one *Staphylococcus aureus,* and one a pneumococcus which was not typed.

DISCUSSION

As already stated, no claim is made that the organism isolated is in every case the agent of the disease. The difficulty of interpretation of the sputum bacteriology has already been fully discussed. But so far as concerns lobar pneumonia we have an indirect check on the general reliability of our results in the comparison of our cases with a parallel series in adults. The bulk of our examinations were made in the first five months of 1921. In Table 4 I have compared the percentage figures for the different types in our series with similar figures for 151 cases in adults admitted to this hospital during the same period.

TABLE 4.—COMPARISON OF INCIDENCE OF PNEUMOCOCCUS TYPES IN LOBAR PNEUMONIA IN ADULTS AND IN CHILDREN

	Adults; Percentage of 151 Cases	Infants and Children; Percentage of 98 Cases
Pneumococcus Type I	37.7	29.6
Pneumococcus Type II (including atypical)	8.0	3.0
Pneumococcus Type III	13.2	7.1
Pneumococcus Group IV	24.5	35.7
Undetermined	16.5	25.7

It will be noted that in adults pneumococcus Type I has been particularly prevalent, that pneumococci of Group IV have been very common, that Type III has occurred with about average frequency, but that there has been a notable paucity of Type II. If we reverse the positions of Type I and Group IV in children, the same order of frequency then follows. More important than that, the actual percentage figures in the two series, type for type, are not so greatly at variance. The parallelism is sufficiently strong to suggest not only that the results we have obtained in our younger patients are, in the main, tolerably accurate, but that the etiology of lobar pneumonia in infants and chil-

dren follows quite closely the etiology of that disease in adults in a given place and season.

It is somewhat more difficult to institute a comparison of our results with those published by other authors for pneumonia in infants and children. For the most part these studies have not attempted to segregate the two varieties of pneumonia—a thing often difficult to do, but from the standpoint both of prognosis and etiology of considerable importance. The more important articles may, however, be reviewed.

Wollstein and Benson,[8] in a series of fifty cases of pneumonia, chiefly in infants, and chiefly lobular, found pneumococcus Type I in 8 per cent., Type II in 12 per cent., Type III in none, and Group IV in 60 per cent. A number of infections were of mixed character.

Mitchell,[9] in a series of ninety cases in infants and children, chiefly lobar, found pneumococci of Group IV in no less than 74.4 per cent. of the cases of lobar pneumonia, which comprised the greater proportion of his cases.

Pisek and Pease,[10] in a series of forty-eight cases of pneumonia in infants and small children, of whom more than half were of the lobar variety, found pneumococcus Type I in 15.2 per cent., Type II in 20 per cent., Type III in 5.6 per cent., and Group IV in 26.7 per cent. The bacteriology of the remaining 32.5 per cent. could not be determined. These figures, though of so small a series, accord more nearly with our own, and with the figures usual in adults, than do the others.

If the figures of these authors be thrown together, it will be found that of 188 cases of lobar and lobular pneumonia, 62.5 per cent. appear to be associated with pneumococci of Group IV, while only 25 per cent. showed the presence of fixed types. Applying the same process to our own 165 cases of pneumonia and postpneumonic empyema, it will be seen that pneumococci of Group IV were present in 33.3 per cent., while 34.6 per cent. showed the presence of fixed types. The approximate ratios of fixed types to heterogeneous strains in the two series are respectively 1 : 2.5 and 1 : 1.

Our studies suggest, then, that in pneumonia in infants and children—in particular the lobar form—the fixed types of pneumococcus are relatively much more common than we have been led to believe. That these small patients are at least potentially more important as carriers of disease than has been thought, follows in course. Indeed, several concrete illustrations of this point may be adduced. Thus, Case 131 is the sister of Case 107; they were admitted within a short time of each other, both suffering from lobar pneumonia caused by pneumo-

8. Wollstein and Benson: Am. J. Dis. Child. **12**:254 (Sept.) 1916.
9. Mitchell: Penn. M. J. **20**:343, 1917.
10. Pisek and Pease: Am. J. M. Sc. **151**:14, 1916.

coccus Type I. A third child is reported to have died at home with pneumonia between the times of the two admissions. Case 98 was the original case in his family; he was admitted with lobar pneumonia, pneumococcus Type I. A few days later his brother, a young adult, was admitted with the same disease, caused by the same type. Case 112 was in the hospital at the same time as her father; both suffered from lobar pneumonia caused by the same type of pneumococcus. Not many illustrations of this sort are needed to establish the point that the same rigid precautions should be carried out with pneumonia in childhood as in adults.

It appears that in general the child has a better natural immunity to his infecting type of pneumococcus than does the average adult. This is well shown in Table 5. From the few positive blood cultures we have obtained it is unfair to draw conclusions; it is suggested, however, that invasion of the blood is of less serious import in the child than in the adult.

TABLE 5.—Comparative Mortality for Lobar Pneumonia in Adults and Children According to Types of Pneumococci

Type	Percentage Mortality, Adults	Percentage Mortality, Children
Pneumococcus Type I	30.0	7.9
Pneumococcus Type II	16.6	0
Pneumococcus Type III	75.0	0
Pneumococcus Type IV	10.8	5.7

It should be noted that type for type, with one exception, there is a higher mortality recorded for bronchopneumonia than for lobar pneumonia (Figs. 1 and 2). This exception is pneumococcus Type III. Although it was found in nine lobar or lobular pneumonia cases, there were no deaths, a fact of exceptional interest, since the death rate for this type in adults has been extremely high. Doubtless, the generally higher mortality in bronchopneumonia is largely due to the conditions of origin and circumstances of that disease, which in the cases studied has been very rarely primary.

Streptococcus haemolyticus and *Staphylococcus aureus* appear as important agents in the bronchopneumonias which we have studied. Responsibility for this disease is charged to the former in 15.4 per cent., and to the latter in 9.6 per cent. The death rates for the two were 87.5 and 80 per cent., respectively. Hence, the occurrence of either of these organisms would appear to be of exceptionally grave prognostic significance. The tendency of *Streptococcus haemolyticus* to produce empyema has been clearly evident (four cases). Recent literature, however, has so repeatedly noted this fact that it is hardly necessary to stress its importance in this place.

I desire to place especial emphasis, however, on one fact developed in these studies for which I can find no figures to correspond in the literature. I refer to the large number of empyemas in these infants and children due to pneumococcus Type I. Of thirty-two cases of pneumonia charged to this type, 12 or 37.8 per cent. developed empyema. The incidence of empyema in the parallel series of Type I pneumonias in adults was 10.5 per cent. Of fifteen cases of postpneumonic empyema in children, admitted as such, eleven were caused by pneumococcus Type I. This organism has been responsible for 72 per cent. of the thirty-two cases of empyema which we have studied. In these infants and children there appears to have been almost as great a percentage liability to empyema from pneumococcus Type I, as from *Streptococcus haemolyticus,* while numerically the former has been much more important. If further studies establish that the application of these facts is permanent and general, not merely seasonal and local, data of considerable prognostic import will have been furnished for pneumonia in children.

Wherein lies the explanation of this exceptional liability to empyema in infections with Type I pneumococcus? It has already been suggested that the child appears, in general, to possess a natural immunity against pneumococcus superior to that of the average adult; witness the comparative mortality, type for type, in the parallel series of cases. On theoretical grounds, at least, it might be susceptible of argument that this liability to empyema is even an actual expression of such superior immunity. For in an individual susceptible to infection at all, the higher the degree of his immunity, natural or acquired, the more likely is the infection to be focal and not general. But the child's naturally superior immunity appears to extend to all types of pneumococcus, whereas it is only in infection with the one type that this extraordinary liability to empyema has been shown. Nor—and this is most important—is there any factor of artificial immunity to be reckoned with in these cases, for in none of them was serum administered. Of three children who received serum none developed empyema. In view of these facts, it seems more reasonable to explain this tendency to empyema with pneumococcus Type I, principally on the basis of some specific inherent property in the organism itself, or to some proper modus operandi. The smaller remaining factor will be some physical or anatomic factor in the child—this quite nonspecific—which renders him generally more subject to empyema than the adult.

These results have even a possible bearing on the treatment of Type I pneumonia in adults. There apparently exists in some quarters an opinion, however ill-grounded, that serum treatment predisposes to the development of empyema. The total independence of this phenomenon from specific therapy in these cases should go some way to

remove this superstition. Moreover, in six cases of Type I empyema following pneumonia in the adult series quoted, four developed in untreated cases. One wonders if even the reverse opinion may not be true—that the early and vigorous use of serum might not actually prevent this untoward development in many cases.

SUMMARY

1. Ninety-eight cases of lobar pneumonia, fifty-two cases of bronchopneumonia and fifteen cases of postpneumonic empyema have been studied bacteriologically in patients 12 years of age or younger.

2. In lobar pneumonia, or empyema following, pneumococcus Type I has been found in 29.9 per cent.; pneumococcus Type II (including atypical) in 3 per cent.; pneumococcus Type III in 7.1 per cent., and pneumococci of Group IV in 37.7 per cent. These figures represent a higher percentage of fixed types than the perusal of a rather meagre literature would have suggested.

3. The general reliability of these findings is suggested by the reasonably close correspondence to the findings in a parallel series of cases in adults. It is further suggested that the etiology of lobar pneumonia in infants and children follows in a general way that in adults for a given community at a given time.

4. Studies of comparative mortality suggest that the child possesses a better natural immunity against pneumococcus, type for type, than does the average adult.

5. In bronchopneumonia the fixed types of pneumococcus are much less common than in lobar pneumonia. Common mouth forms are relatively much more common. Regardless of type, the general mortality for bronchopneumonia is much greater than for lobar pneumonia. This is probably due to the conditions of origin and circumstances surrounding the former disease. *Streptococcus haemolyticus* and *Staphylococcus aureus* have been common in bronchopneumonia and have been followed by a particularly high mortality.

6. There has been an extraordinary tendency to the development of empyema in infections with pneumococcus Type I. Nearly 38 per cent. of pneumonias charged to this type developed this complication. Numerically it outranks any other cause of empyema in this series by about six times. This phenomenon bears no relation to the administration of serum.

It is hoped that further studies may be undertaken to determine the general applicability of these results.

CLINICAL DEPARTMENT

CONGENITAL TUBERCULOSIS WITH REPORT OF CASE *

GEORGE P. PRATT, B.Sc., M.D.
OMAHA

Pehu and Chalier (1908, 1914 and 1915) from an extensive study of congenital tuberculosis accept only fifty-one cases as definitely proven. In the analysis of these cases they conclude that the father plays no part in the transmission, all the cases being the result of transplacental "heredocontagion." In all the cases admitted, the mothers had grave exacerbations of tuberculosis and they regard it as plausible that there was an acute generalization of the disease.

However, Weller (1916) and Warthin (1913) report cases of miliary tuberculosis of the placenta with latent tuberculosis in mothers, and Grulee and Harms (1915) report the case of a child succumbing on the eleventh day with generalized miliary tuberculosis whose mother had healed tuberculosis of the hip joint. Weber in a survey of congenital tuberculosis (1916) states: "It seems to me that we may well suppose that occasionally a mother with some form of chronic, and perhaps even latent tuberculosis, may suffer from a temporary condition of tuberculous 'bacillemia' during pregnancy, without succumbing; for, probably conditions of temporary tuberculous 'bacillemia' are not exceedingly rare in the subjects of chronic tuberculosis, apart from pregnancy."

These latter views are mentioned merely as possibilities and, perhaps, to arouse interest in this subject. The case which I am reporting conforms rigidly to the concepts of Pehu and Chalier.

The condition in the foetus may be that of a true tuberculosis in which macroscopic and microscopic lesions are present or the status bacillaris described by J. Honl (1894) in which the tubercle bacilli can be found in the foetal organs, unaccompanied by macroscopic and microscopic changes.

REPORT OF CASE

CASE (No. 5290).—*History.*—A Mexican woman, married, aged 24, was admitted at the University Hospital, Jan. 6, 1921, in a critical condition, delirious, markedly cyanotic, breathing rapidly and perspiring freely. She complained

* Received for publication, Aug. 15, 1921.

* From the Medical Department of the University of Nebraska College of Medicine.

of pain in the chest, cough, headache and diarrhea. The first illness began one month ago with weakness, pain in the chest, cough, diarrhea and vomiting, the vomiting occurring after meals. The above symptoms gradually grew worse until two weeks prior to admission when she was confined to her bed. A physician called at this time, made a diagnosis of pneumonia. The symptoms have continued and increased since the onset.

Physical Examination.—The breasts were increased in size, pigmented and colostrum could readily be expressed from both nipples. The apex beat was located in the fifth interspace just inside the midclavicular line. No cardiac murmurs were heard and the second pulmonic sound was accentuated. The pulse was regular but weak. There was impaired resonance over the right base anteriorly and over both bases and apices posteriorly. Large and small bubbling râles and musical râles could be heard over the entire chest, both on inspiration and expiration. A slight friction was noted in the left post-axillary line. The abdominal wall on palpation was relaxed. The liver could be felt two fingers breadth below the costal margin and was slightly tender. The lower abdomen was filled with a palpable rounded mass extending to one inch below the umbilicus. A vaginal examination and the fetal heart tones confirmed a pregnant uterus.

The temperature dropped from 103 to 99 F. on the second day in the hospital and remitted then from 101 to 102 F. or 103 F. The respirations were always above 36 per minute, perhaps an average of 45. The pulse averaged between 115 and 130 until the end. The blood examination gave negative Widal reactions, negative blood cultures, a count of 5,600 leukocytes and a one plus Wassermann. At no time could tubercle bacilli be demonstrated in the scanty sputum. The urine was negative, except for a strongly positive diazo reaction.

Diagnosis.—The condition was diagnosed clinically as acute miliary tuberculosis and pregnancy. Death occurred Jan. 10, 1921.

Necropsy Report.—The necropsy revealed a generalized distribution of characteristic miliary tubercles in all the organs but most numerous in the lungs, making the case a predominant pulmonary form of miliary tuberculosis. The uterus was markedly enlarged, soft, congested and corresponded in size to that of five months pregnancy.

In order to facilitate the examination to determine the possibility of placental and fetal tuberculosis, the entire uterus with the fetus in situ was fixed in 20 per cent. liquor formaldehyd solution over night. The following morning the uterine cavity was opened and the fetus and placenta left exposed for forty-eight hours for complete fixation after which the abdominal cavity of the fetus was opened under aseptic precautions and free from contact with placenta or uterus. A distinct increase of straw-colored fluid was found in the fetal peritoneal cavity. Smears of this were negative for the bacillus of tuberculosis. No evidence of miliary tubercles was found in the abdominal cavity. The liver was somewhat enlarged and section was made of it in a dry field under aseptic precautions. Smears were made of scrapings from the cut surface and in these smears occasionally tubercle bacilli were found with Ziehl-Neelsen's carbolfuchsin stain. Similar smears were made from the placental cut surface before complete fixation, and prolonged search of these failed to reveal any tubercle bacilli.

The microscopic sections of the fetal liver, heart, lungs, spleen, kidney and intestines showed no tubercles and no thrombotic lesions. The microscopic sections of the placenta showed no tubercles and tubercle bacilli could not be demonstrated by stain and prolonged search.

Pathologic Diagnosis.—Miliary tuberculosis with predominant pulmonary localization; pregnancy; fetal tuberculosis without gross or microscopic lesions.

The finding of the tubercle bacilli in the fetal liver establishes this as a proven case of congenital tuberculosis which conforms to the status bacillaris of J. Honl, since no lesions of tuberculosis could be found after a careful microscopic examination of the fetal organs.

The most striking feature in the study is the difficulty of finding grossly or microscopically, pathologic evidence of tuberculosis or the tubercle bacillus in the placenta or fetus. It may be on account of this difficulty and because a thorough examination of placental tissue is not made routinely that we are lacking in the proof that tuberculosis may be congenitally transmitted from a mother with latent tuberculosis to children who develop the disease in the early months of life.

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CONTENTS

PAGE

The Nature of the Plantar Reflex in Early Life and the Causes of Its Variation. W. M. Feldman, M.D., B.S., London 1

Can Yeast Be Used as a Source of the Antineuritic Vitamin in Infant Feeding? Amy L. Daniels, Ph.D., Iowa City 41

The Incidence of Protein Sensitization in the Normal Child. M. Murray Peshkin, M.D., and William L. Rost, M.D., New York 51

An Unusual Exanthem Occurring in Infants. Roy M. Greenthal, M.D., Ann Arbor, Mich. 63

The Respiratory Exchange in a Case of Biliary Atresia. G. B. Fleming, Glasgow, Scotland 66

Bacteriologic Studies of One Hundred and Sixty-Five Cases of Pneumonia and Postpneumonic Empyema in Infants and Children. Arthur Bates Lyon, M.D., Boston 72

Clinical Department

Congenital Tuberculosis with Report of Case. George P. Pratt, B.Sc., M. D., Omaha 88